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SURGERY

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SURGERY

VOL. 38 DECEMBER, 1955 No. 6

Original Communications

Society for Vascular Surgery
(Concluded)

TREATMENT OF ARTERIOSCLEROTIC OCCLUSIVE DISEASE OF THE LOWER EXTREMITIES BY EXCISION AND GRAFT REPLACEMENT OR BY-PASS

E. STANLEY CRAWFORD, M.D., OSCAR CREECH, JR., M.D., DENTON A. COOLEY, M.D., AND MICHAEL E. DEBAKEY, M.D., HOUSTON, TEX.

(From the Department of Surgery, Baylor University College of Medicine, and the Methodist, Jefferson Davis, and Veterans Administration Hospitals)

THE obstructing arteriosclerotic lesion producing arterial insufficiency of the lower extremity is located below the aortic bifurcation in the majority of patients and most commonly in the superficial femoral artery.[8, 11] Moreover, the occlusive process is frequently segmental in nature with the vessel having a relatively normal lumen above and below it.[10] As a consequence of this fortunate pathologic feature of the disease, it has been possible to restore circulation toward normal levels in the peripheral arterial bed by direct surgical methods.

Although numerous technical variations are possible, there are essentially two methods of direct attack. The first of these to be employed was thromboendarterectomy as originally described by dos Santos[5] and subsequently advocated by others.[1, 6, 12] The percentage of successful results following this method has varied considerably, but in most reports the incidence of both early and late failures has been high.[1, 5, 6, 9, 12] The second and more recent approach consists in the substitution of another blood vessel for the obstructed segment.[6, 7, 9, 13, 14] This may be performed by one of two methods, namely, excision of the occluded segment and its replacement with a blood vessel graft or using the graft to by-pass the obstructed segment without removing it. Numerous reports indicate that success with this approach is more consistent and

Supported in part by grants from the Houston District Chapter, Texas Heart Association, and the Cora and Webb Mading Fund for Surgical Research.

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

superior to that of thromboendarterectomy, especially in patients with long-diseased segments.[6, 7, 9, 13, 14] It would also appear that greater success is obtained from the use of an artery than a vein graft.[14] The purpose of this communication is to report our experience with these methods using lyophilized arterial homografts in the treatment of 63 patients with occlusive lesions located below the aortic bifurcation and to indicate the advantages of the by-pass procedure.

CLINICAL MATERIAL

All patients entering the hospital with arterial insufficiency of the lower extremity associated with absent femoral or popliteal pulses are investigated with arteriography, and those who show a patent artery of reasonable size below the obstruction and above the bifurcation of the popliteal artery are selected for operation irrespective of the distance of the occlusive process above this level. While the proximal extent of the occlusion is of little consequence, an essential requirement for operation is the presence of a good outflow in the peripheral arterial bed. Accordingly, it is important to ascertain patency of one or more of the distal branches of the popliteal artery. The presence of secondary blocks below this level does not appear to alter the results significantly and, therefore, does not contraindicate operation.

Slightly over 50 per cent of the patients with femoral occlusions and practically all patients with iliac occlusions surveyed in this manner have been candidates for operation and almost all of them have had grafts inserted. The few patients with operable lesions not subjected to operation had advanced impairment of cardiac or renal disease and the associated intermittent claudication was not considered sufficient indication to justify the risk of surgical intervention. Eight patients had diabetes and 6 had had previous myocardial infarction. The ages of the patients ranged from 34 to 72 years, with the average being about 50 years.

The 63 cases in this series are divided primarily into two groups on the basis of the technique by which the graft was inserted, but also analyzed according to the site of occlusion. The end-to-side by-pass procedure was used in 40 extremities, 16 of which had iliac and 24 femoral artery occlusions. Excision of the diseased segment with graft replacement by end-to-end anastomosis was carried out in 23 extremities, 12 of which had femoral and 11 iliac artery occlusions. In addition, but excluded from this series, there are 85 cases of occlusion of the iliac artery associated with the Leriche syndrome or thrombo-obliterative disease of the terminal aorta and bifurcation. Under these circumstances a part of the iliac artery is resected, and the short remaining occluded segment has been endarterectomized. The bifurcation aortic homograft has been anastomosed to this endarterectomized distal iliac segment with highly gratifying results.[4]

The indication for operation was intermittent claudication in 54, gangrene of limited degree in 8, and in 1 patient extensive gangrene. Gangrene was associated with painful ischemic ulcers in 2, severe rest pain in 7, and in 1 a digit

had been destroyed by associated infection. Operation was advised in the patient with extensive gangrene to permit a low-leg amputation since the opposite leg had been amputated previously at a higher level.

TECHNIQUE OF OPERATION

Since the objective of the operation is to restore a normal pulsatile blood flow into the peripheral arterial bed, it is essential to bridge the entire occluded segment, including all areas of proximal or distal narrowing of the vessel. In patients in which the occlusive process is relatively short and discrete, excision and graft replacement may be used (Fig. 1), but in larger occlusions or those with partial stenosis well above and below the area of complete

A. *B.*

Fig. 1.—*A*, Arteriogram of left superficial femoral artery showing localized area of arterial occlusion. The specimen superimposed on the arteriogram consists of the excised occluded segment of femoral artery together with some occluding thrombi removed from the proximal narrowed portion by endarterectomy. *B*, Arteriogram of the same artery made ten days after excision of the occluded segment and insertion of an arterial homograft.

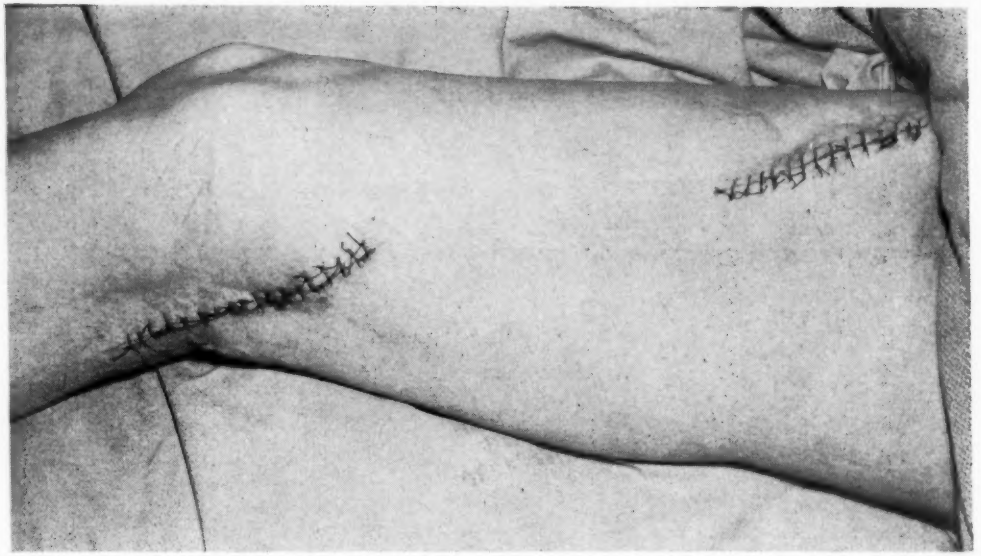
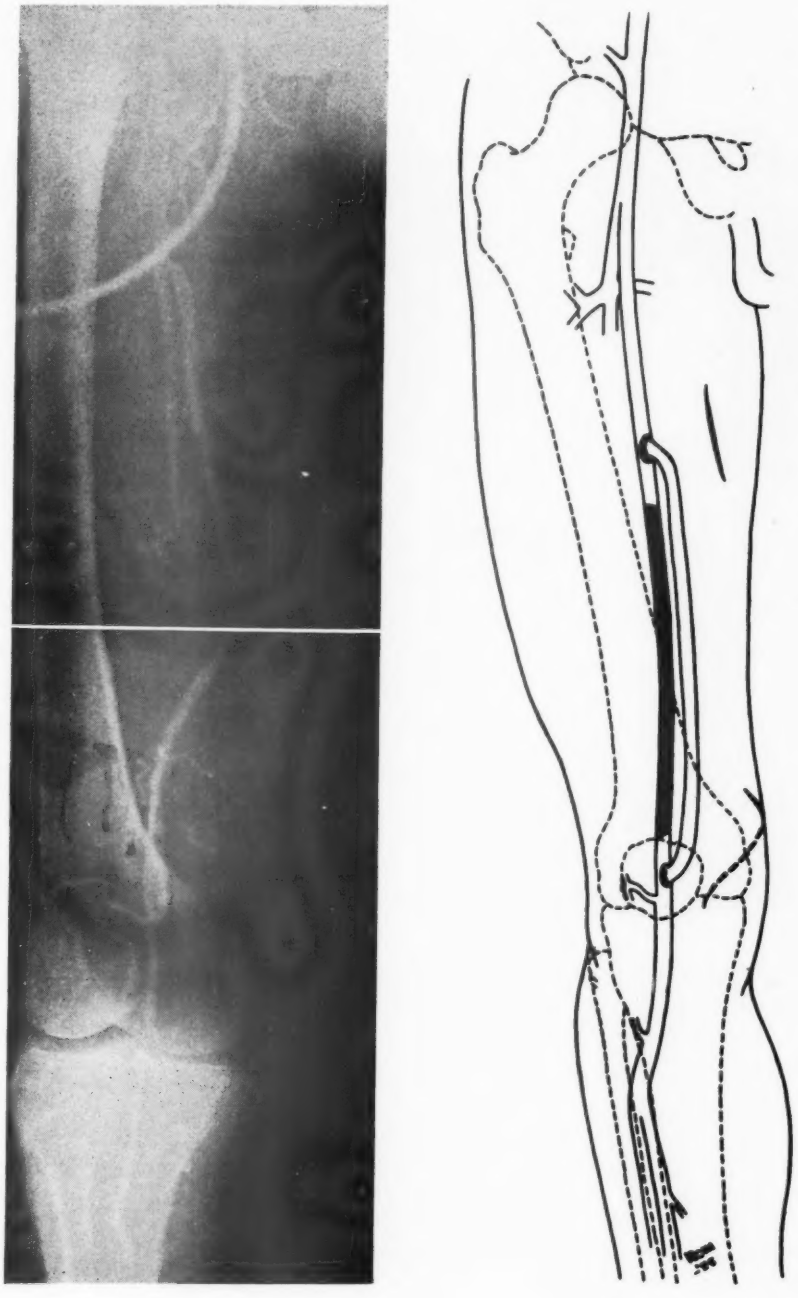


Fig. 2.—Photograph of right lower extremity showing location of incisions used by-pass a relatively long occluded segment of superficial femoral artery.

Fig. 3.—Photographs made at operation in same case shown in Fig. 2 showing *A*, the completed end-to-side anastomosis between the arterial homograft and popliteal artery, and *B*, a similar anastomosis between the proximal end of the homograft and superficial femoral artery.

obstruction, the by-pass procedure is preferable (Figs. 2 to 4). Thus, in 23 extremities in this series the occlusive lesions were less than 15 cm. and these were completely excised and replaced with arterial homografts. In the other 40 patients with relatively long or multiple obstructions, the by-pass procedure was employed as originally proposed by Kunlin and associates.[9] This is accomplished by performing an end-to-side anastomosis of the graft to



A. *B.*

Fig. 4.—Arteriogram (*A*) and explanatory drawing (*B*) showing satisfactory restoration of circulation in popliteal artery after insertion of by-pass arterial homograft and undisturbed collateral network of vessels.

the host artery above and below the occluded segment through two small separate incisions which are connected by a tunnel made by blunt dissection through Hunter's canal using a vein stripper or a ring-tipped sponge clamp. Although homologous vein grafts were used by Kunlin and his workers,[9] we have found freeze-dried arterial homografts preferable for several reasons.[3] For one thing, it simplifies the procedure by eliminating that part of the operation required in obtaining a vein graft and thus reduces the extent of tissue dissection and possible wound complications. For another, it is often impossible to obtain a vein graft of sufficient length and uniform lumen size to bridge the distance required, particularly in cases with relatively long occlusions. It has been necessary, for example, in some instances to extend the graft from the aorta just above the bifurcation to the popliteal artery.

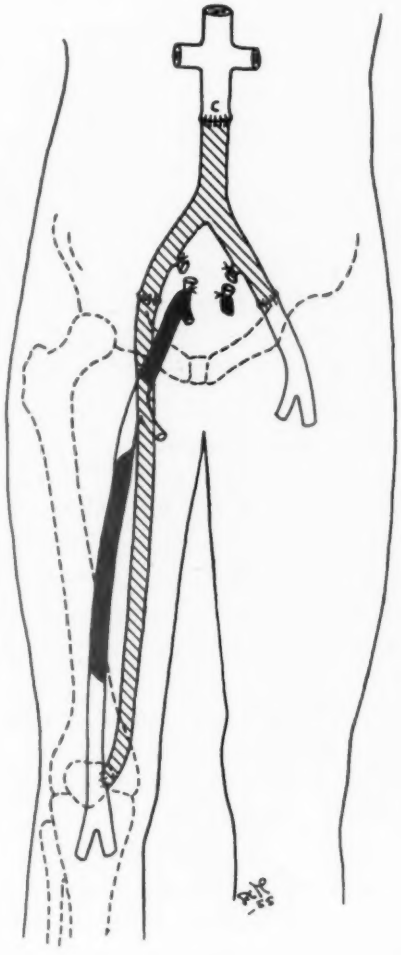
A. *B.*

Fig. 5.—Aortogram (*A*) and femeropopliteal arteriogram (*B*) showing extensive obliterative changes involving the terminal aorta, iliac, common, and superficial femoral arteries. *C*, Drawing showing method of restoration of circulation to the leg by use of aortic and iliofemoral arterial homografts.

Technical considerations of the by-pass procedure have been presented in detail elsewhere.[2] It consists essentially in making two small incisions, one above and the other below the obstruction, placed at the proper levels to expose relatively normal segments of the artery as determined by the arteriogram. The two incisions are connected by a tunnel which is made by blunt dissection with the index finger and completed with a vein stripper or ring clamp. Following exposure of the artery and the application of atraumatic arterial clamps, an oval aperture is made in its wall approximating the size of the lumen of the graft and placed in the long axis of the artery. The end of the arterial homograft is then anastomosed to this opening in the host's artery as an end-to-side anastomosis, using a continuous suture of No. 5-0 arterial silk. After completing the anastomosis above, the free end of the graft is drawn through the tunnel and anastomosed in a similar manner to the segment of artery exposed in the lower incision.

RESULTS

Successful restoration of blood flow in the peripheral arterial bed distal to the occlusion following operation was determined by arteriography or by the presence of palpable pulses below the occlusion not felt before operation. All iliac grafts remained patent following discharge from the hospital irrespective of the technique employed. In the femoral group, however, failures occurred in 3 of 24 by-pass grafts and in 2 of 12 in which the occluded segment was



C

Fig. 5, *C*. *(For legend see opposite page.)*

excised and a graft inserted by end-to-end anastomosis. The failures in the former group were immediate and were attributed to inadequate peripheral outflow bed. In all of these cases the by-pass graft was anastomosed to popliteal segments that were too small and without adequate patent branches. The possibility of failure in these cases was anticipated but operation was attempted in the hope that some increase in blood flow could be obtained. These cases

must now be regarded as errors in selection. Particularly significant is the fact that none of these cases in which the by-pass procedure was employed was made worse by the operation.

The indication for operation in the 5 patients in whom a graft procedure failed to restore blood flow distal to an occlusion was intermittent claudication. The clinical condition and the arterial circulation of the 3 extremities in which a by-pass graft was attempted remained unchanged. The clinical condition of 1 extremity in which the occlusion was excised and a graft inserted by end-to-end anastomosis also remained unchanged. In the second case, however, in which this technique was unsuccessfully employed, the arterial circulation was severely impaired and amputation was required in the ensuing two weeks.

The clinical success in those extremities in which the graft remained patent was manifested by striking improvement in circulation. Intermittent claudication, rest pain, and ischemic ulcer pain were completely and immediately relieved. The two ischemic ulcers rapidly healed and the mild gangrenous changes quickly disappeared. An open amputation was performed on the infected toe and this wound healed rapidly. The patient with extensive gangrene preoperatively had a necrotic foot with color demarcation at the mid-tibial level, and coolness above the knee. Following a by-pass graft, pulses were felt distal to the block and normal circulation was evident at the ankle. A low-leg amputation was successfully performed and the additional length of this stump has been a definite asset in the rehabilitation of this patient in view of the previous amputation of the opposite leg.

All patients have been carefully followed since discharge and, in the majority of cases, for periods ranging from six months to little less than two years. In all of them the grafts have remained patent and no patient has had recurrence of symptoms. Successful results were thus obtained in all of the 27 cases with occlusion of the iliac artery and in 31 (86 per cent) of the 36 cases with occlusion of the femoral artery.

The longest graft inserted extended from 3 cm. below the renal arteries to the popliteal artery on one side and just proximal to the inguinal ligament on the other (Fig. 5). A bifurcation aortic homograft replaced a partially occluded distal aorta which thrombosed after endarterectomy in the early stages of the operation. A long iliofemoral arterial homograft was anastomosed to the external iliac branch of this graft and used to by-pass a total occlusion of the right common and external iliac arteries in the pelvis and a second occlusion of the superficial femoral artery. Pedal pulses not present before operation were immediately palpated after operation and the patient has been relieved of intermittent claudication.

COMPLICATIONS

There were no deaths in this series and few complications. One patient developed a superficial wound infection and another had thrombophlebitis associated with a small pulmonary infarct but both recovered completely without residual disturbances. No patient has had saphenous nerve neuralgia or other symptoms of nerve damage. The single major amputation performed following

a by-pass graft was for a part that had been destroyed prior to hospital admission. The preoperative circulation was unaltered in the 3 failures using this technique. In the excision and graft group, the circulation of 1 extremity was severely impaired by an unsuccessful operation and low-thigh amputation was required.

DISCUSSION

It is our opinion that the relatively good results obtained in these cases were due to proper selection of patients, the use of homologous arterial grafts, and, particularly, to the simplified technique of the end-to-side by-pass procedure in the majority of the operations. As indicated previously, patients were selected on the basis of the arteriographic findings, as well as on their general ability to withstand the operation.

In all cases consideration was given to the over-all value to the patient of a gain in peripheral circulation. The decision was easy in the patient who had a lesion that threatened loss of an extremity, since these patients withstood the graft operation which preserved the extremity more easily than an amputation. The patient who has significant cardiorenal disease and intermittent claudication as the only indication for operation has been refused operation, not only because of increased risk but also because there was little or no need for increased blood flow into the extremity to permit extra activity. Perfect results based upon patency of the graft are possible by conservative selection from the arteriogram if one operates only upon those with definitely large patent distal segments. Several candidates that were thought to have been border line were successfully grafted using the by-pass technique and have received the benefits of this improved form of treatment. The fact that among all cases studied all of those with iliac occlusions were successfully grafted and somewhat over half of those with femoral occlusions were found to be candidates for operation with successful results obtained in 86 per cent, comparing favorably with other reports,[14] indicates the wide application of this form of treatment.

Arterial grafts of desired length have been available as needed and are prepared as the patient is readied for operation.[3] This has ensured an adequate vessel to be used as a graft and has spared the extremity of additional operating time and dissection. The uniform caliber of the arteries of great length used in most of these cases has ensured a good, vigorous blood flow and was undoubtedly responsible for the absence of thrombosis. The low incidence of wound complications and leg edema is probably related to the minimized dissection and injury by not using the patient's own veins.

The end-to-side by-pass operation has been found technically far more satisfactory in avoiding the factors of direct surgery which contribute to failure than any other procedure in our experience. It is based upon the natural response of the arterial bed to occlusion, which is the development of collateral vessels around the area of obstruction (Figs. 4, *A* and 6). Accordingly, the operation consists essentially in the addition of a collateral around the diseased segment equal in size to the host artery. The two small incisions used require minimal dissection, thereby reducing injury to neighboring structures such as

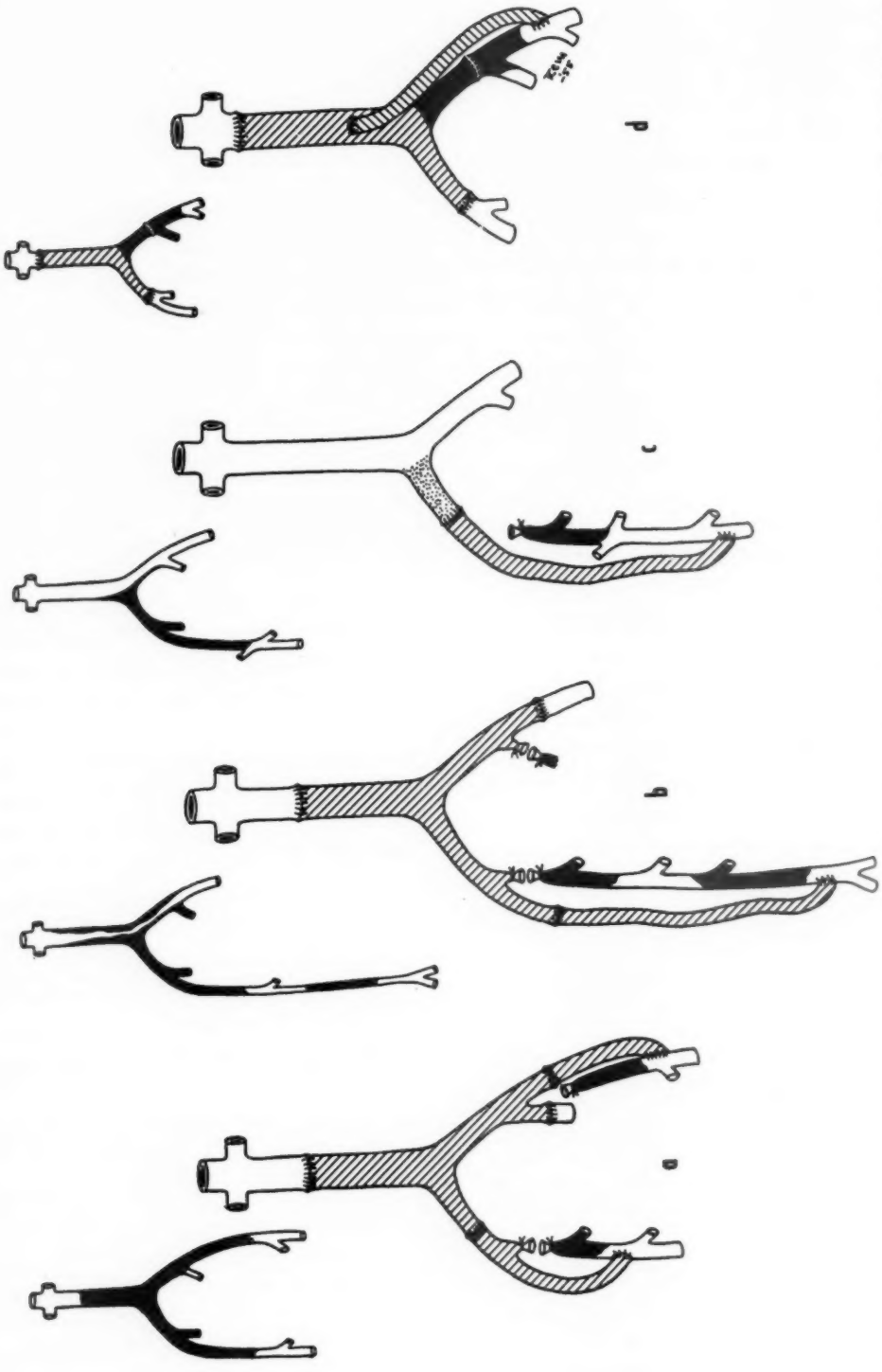


Fig. 6.—*a-d*, Diagram showing various types of aortic and major arterial occlusion and the method of restoring circulation by utilizing by-pass procedure employing arterial homografts.

nerves, veins, and lymphatics. Troublesome and even incapacitating complications are prevented. The collateral branches are not sacrificed and the most normal segment of artery above and below the obstruction can be safely used for the anastomosis, thereby ensuring the best possible blood flow. The avoidance of injury to existing collaterals prevents postoperative reduction in blood flow should the graft fail. This important feature of the procedure has been a source of reassurance in operations upon borderline candidates.

It would appear significant that all of the 27 patients with occlusion of the iliac artery obtained successful results, whereas the only failures occurred in patients with occlusion of the femoral artery. Of further significance is the fact that the only failures with the by-pass procedure occurred in patients with peripheral extension of the occlusive process producing an inadequate outflow bed. These observations would suggest that the more proximal the segmental occlusive process the greater is the chance of success. It also emphasizes the importance of a good peripheral outflow bed as a criterion for operation.

Lumbar sympathectomy has not been routinely performed on the grafted side unless the attempt to employ the graft procedure proved unsuccessful. This operation is not infrequently performed on the opposite side at the same time of the graft procedure when that extremity is also involved by an occlusive process not associated with a patent lower segment. It is noteworthy that 8 of the patients in this series had had sympathectomies performed previously, 7 for intermittent claudication and 1 for arterial insufficiency associated with infection. The symptom of intermittent claudication was unchanged by this operation in the former 7 patients. The latter patient, who had not had this symptom prior to sympathectomy, developed intermittent claudication three months later. All 8 patients were subsequently relieved of this manifestation by a graft procedure.

SUMMARY

1. Arteriographic studies on patients with chronic arteriosclerotic arterial insufficiency of the lower extremities with absent femoral or popliteal pulses have demonstrated that the occlusive process is segmental in nature with a patent vessel above and below it in over half the cases. As a consequence of this fortunate pathologic feature of the disease, it is possible to restore circulation toward normal levels in most of these patients by direct surgical treatment, consisting essentially in the substitution of another blood vessel, an arterial homograft, for the obstructed segment.

2. This may be performed by one of two methods, namely, excision of the occluded segment and its replacement with a blood vessel graft or using the graft to by-pass the obstructed segment without removing it. The former is employed in cases in which the occlusive process is relatively short and discrete, while the latter is used in those with relatively long obstructed segments. Although the proximal extent of the occlusion is of little consequence in determining operability, an essential requirement for operation is the presence of a good outflow in the peripheral arterial bed.

3. This report is based upon an analysis of 63 cases in which these procedures were employed, 27 of which involved the iliac and 36 the femoral artery. Excision with graft replacement was used in 23 cases, 11 of which involved the iliac and 12 the femoral arteries. The by-pass procedure was employed in 40 cases, 16 of which involved the iliac and 24 the femoral arteries. Completely successful results with restoration of distal pulsatile arterial circulation were obtained in all 27 cases with iliac occlusion and in all but 5 of those with femoral occlusions. Two of the failures occurred among the 12 treated by excision and graft replacement and 1 of these was made worse by the operation, requiring subsequent amputation. The other 3 failures occurred among the 24 cases in which the by-pass procedure was employed and may be attributed to an inadequate peripheral outflow bed, thus constituting an error in selection for operation. Of particular significance, however, is the fact that none of them was made worse by operation.

4. There were no deaths and few complications in this series.

5. The conviction has been gained from this experience that the by-pass procedure, owing to its simplicity in concept and application, is superior to other methods of treatment, particularly in patients with relatively long occlusive segments. It has been found to be not only most effective in achieving the desired objective of restoration of normal pulsatile blood flow into the peripheral arterial bed, but also associated with minimal jeopardy to the existing circulatory and functional capacity of the extremity.

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ARTERIOGENESIS INDUCED BY TUBES OF PLASTIC MESH

LOUIS G. HERRMANN, M.D., AND CLAUDE BOLLACK, M.D.,* CINCINNATI, OHIO

(From the Department of Surgery, College of Medicine, University of Cincinnati, and the Lucie Rawson Laboratory for Vascular Research, Cincinnati General Hospital)

THE great strides which have been made in the field of vascular surgery during the past five years, and which were so ably presented here today, give us all reason to be very proud. We are certain this progress will continue but we would like to pause and reflect for a few minutes upon the pathologic-physiologic aspects of arterial replacement. The recent surgical literature is replete with reports of successful bridging of defects in large arteries by a great variety of methods and with an equally great variety of autogenous, homologous, metallic, and synthetic prostheses. We believe each of us should now direct part of our efforts toward a critical analysis of our own experiences with these various materials, with the main object of learning more about the mechanisms which are responsible for the success or failure of any given prosthesis to continue to fulfill the physiologic function of a large artery when transplanted into man.

Ever since the pioneer work of Payr,[1] Carrel,[2] Watts,[3] Tuffier,[4] Leriche,[5] and Murray,[6] our vascular surgeons have realized the importance of re-establishing the continuity of large arteries after extensive injury. We had accomplished this in occasional patients in whom the injury to large arteries resulted from extensive wounds of violence; nevertheless, it was not until the urgencies of war made available everywhere the effective antibiotic agents, the means for effective supportive treatment during long surgical operations, improved anesthesiology, and more suitable instruments and materials, that the routine re-establishment of large arterial pathways could be performed safely and with excellent chances of lasting success, as we heard this morning from our president, Dr. Linton.

We have all been aware of the fact that autogenous materials are most desirable, but no single tissue or sacrificable blood vessel in man has been wholly satisfactory as a replacement for a large artery. Tubes made from peritoneum or fibrous tissue, and even large veins, lack the tough elastic layer of a large artery. Dilatation or degeneration, with eventual occlusion by thrombosis or fibrosis, was the usual end result of such free vascular grafts, although a small percentage of these grafts did prove successful for many years.[6]

The bridging of defects in large arteries, especially those arteries in which there is great intraluminal pressure, presents other problems and hazards. In order to provide additional strength in a vascular graft, surgeons again began

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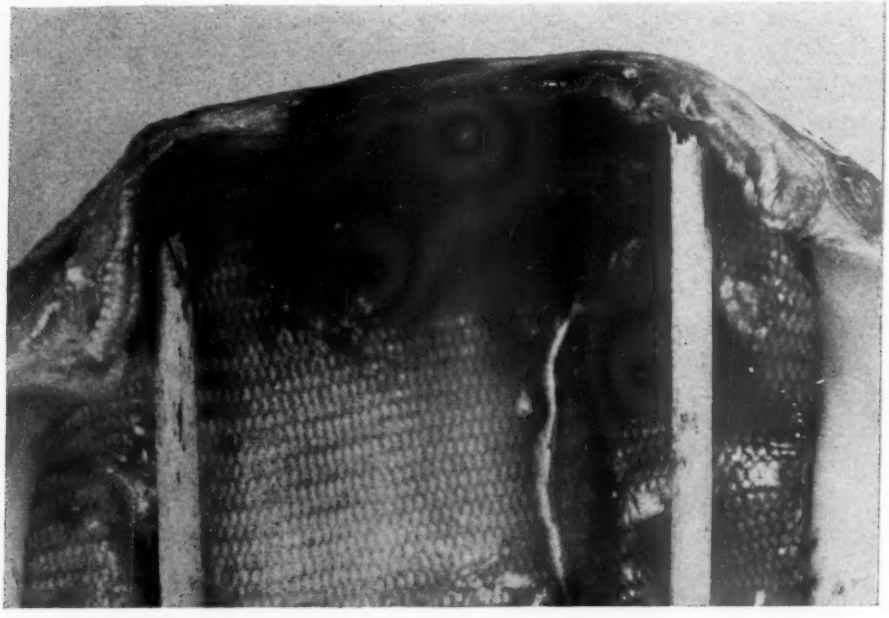
*Fellow of the Rockefeller Foundation from the University of Strasbourg (Surgical Service of Professor Fontaine), France, 1954-1955.

to implant fresh or preserved segments of large arteries from other human beings, even though past experience had already shown that homologous grafts of all living tissues of man failed to retain their original function after transplantation. The technical, biologic, and legal aspects incident to obtaining, sterilizing, storing, and implanting such homologous arterial grafts present further obstacles to their universal use.

A.



B.

Fig. 1.—*A*, Photograph of the adventitial layer of fibrous tissue which has been laid down about the plastic mesh tube 30 days after implantation of the prosthesis; *B*, enlarged view of the wall of the newly formed artery, showing the smooth, glistening, intimal-like lining of fibroblasts and the thick adventitial layer of fibrous tissue with the plastic mesh serving as the elastic tissue layer of the aorta.

It was only natural for investigators to study again the fundamental aspects of the problem and renew the search for a more suitable and easily obtainable substance to bridge defects in large arteries. A great variety of synthetic materials were being produced by the chemists and some of the sheets and fibers even surpass the strength, smoothness, and inertness of silk and metals. The body tissues tolerated many of these plastic substances even better than they

tolerated fibers of animal origin. Surgeons therefore began to implant tubes of these impervious plastic materials, with and without a lining of autogenous vein, to bridge defects in large arteries. These tubes proved reasonably successful and, because of their inertness and their blood-repellent property, they functioned, with relatively few complications, as replacements for segments of large arteries of the neck and the extremities.

It has been our belief for many years that the replacement of large arteries is primarily a physiologic rather than an anatomic problem. To fulfill all the physiologic requirements, at the present stage of our knowledge of the antigen-antibody reaction, defects in large arteries must be bridged by autogenous tissues. It therefore seemed logical to try to promote arteriogenesis by the use of an elastic framework of some inert and pervious material, through and about which the fibroblasts could proliferate and form permanent functional, as well as protective, layers for the newly formed artery.

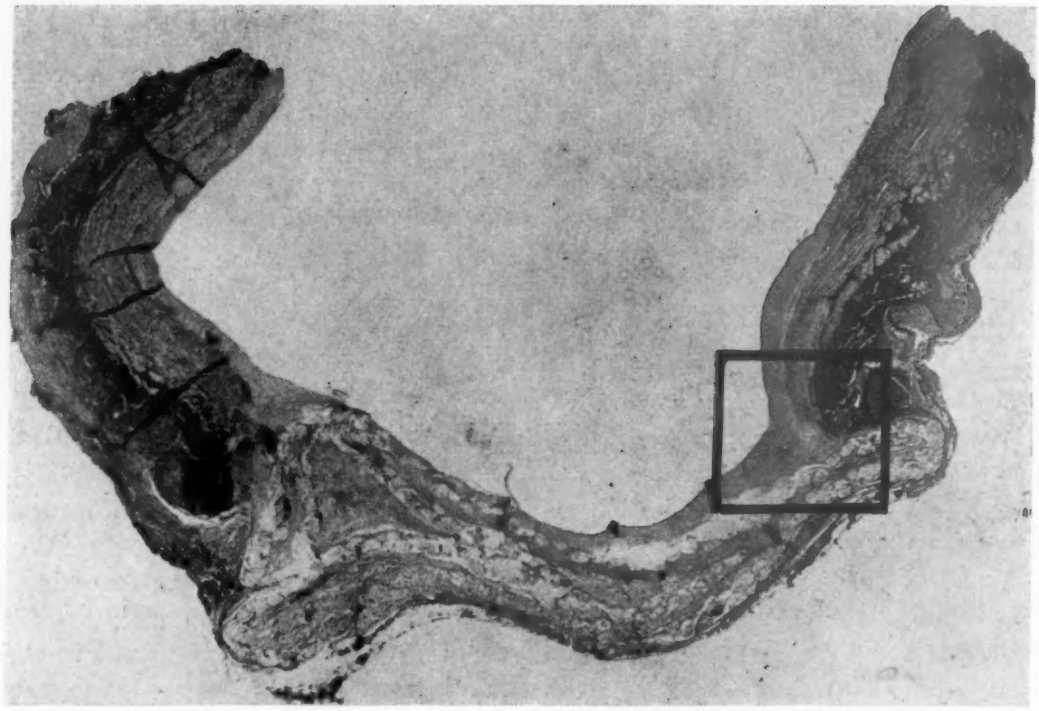
Voorhees, Jaretzki, and Blakemore[7] suggested the first practical approach to the fulfillment of the physiologic requirements for arteriogenesis when they demonstrated that closely woven cloth of Vinyon-N caused the autogenesis of arterial coats from fibroblasts, while the plastic cloth tube actually served as the elastic tissue layer of the newly formed artery. We believe this is a fundamental contribution to the problem of arterial replacement and our own experiences with fine mesh tubes of Orlon and Vinyon-N fibers have convinced us that "arteriogenesis" by this means is not only possible but is a major step toward the practical solution of some of our most perplexing surgical problems.

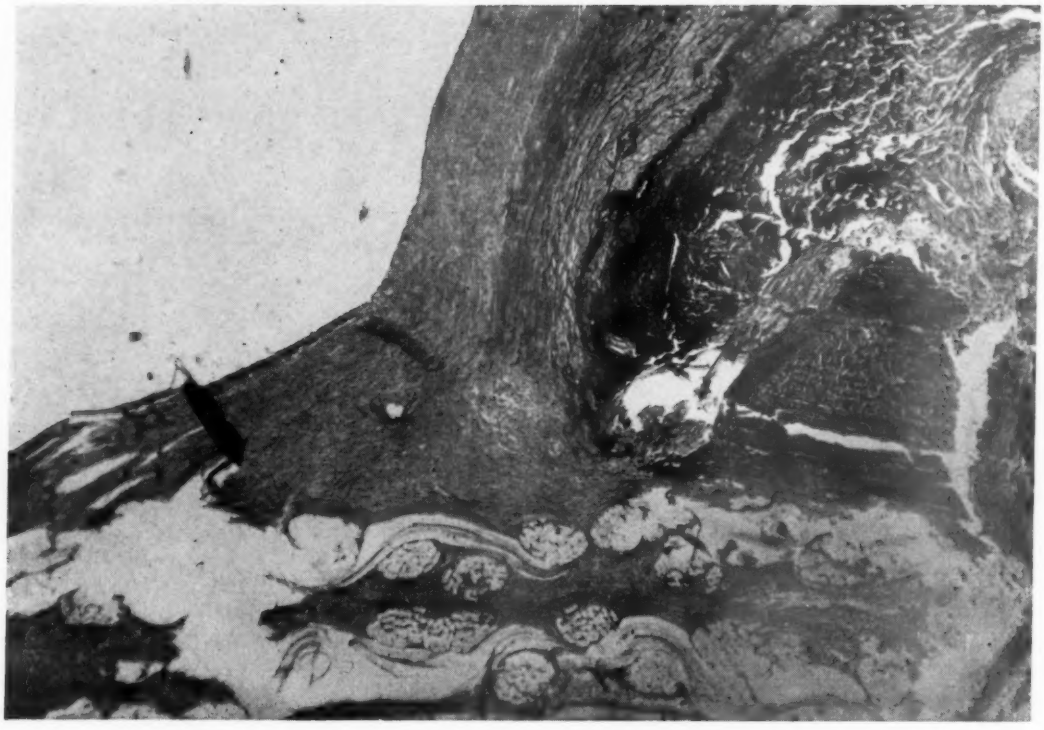
Hufnagel,[8] Shumacker,[9] and their associates used impervious plastic materials and their results have added much to our knowledge of the biologic reaction to these synthetic substances. We believe, however, that the remarkable ability of the plastic cloth to bring about arteriogenesis depends upon the physical property of being pervious as well as elastic.

HISTOLOGIC STUDIES

Histologic sections made at various intervals from two days to one year at the junction of the aorta with the plastic mesh tubes or patches of Orlon or Vinyon-N mesh have been studied carefully. It has been found that the plastic mesh tube is quickly covered by a layer of fibrous tissue which is continuous with the adventitia of the aorta (Fig. 1). There appears to be no cellular reaction around the fibers of the Orlon or Vinyon-N in any of the specimens which have been removed, and only occasional giant cells were found about the fine silk sutures which were used to hold the plastic mesh tube in place at the original operation (Fig. 2). The inside lining of the plastic mesh tube is made up of a thin layer of cells which are of fibroblastic origin. These fibroblasts are seen to extend through the meshes of the plastic tube from the outer layers of the artery and the fibrous tissue layer which is continuous with the adventitia of the artery. Within thirty days, a well-formed layer of fibroblasts lines the entire plastic mesh tube and this layer is continuous with the intimal lining of the

A.





B.

Fig. 2.—*A*, Cross-section of an aorta upon which a patch of Vinyon-N mesh had been sewn to repair a defect. The degree of autogenesis of the layers of the artery is shown 97 days after the patch was sewn on the aorta; *B*, enlargement of the small square shown in *A*, demonstrating that the intimal layer of the normal artery is continuous with the newly formed layer of fibroblasts which forms the inside covering of the plastic mesh patch.

aorta above and below the prosthesis (Fig. 3). The blood-repellent property of these plastic fibers is probably responsible for the fact that thrombosis does not take place before the proliferation of the fibroblasts from the outside of the tube to the inside is complete. In our experience, thrombosis did not occur in any of the plastic mesh tubes which were implanted.

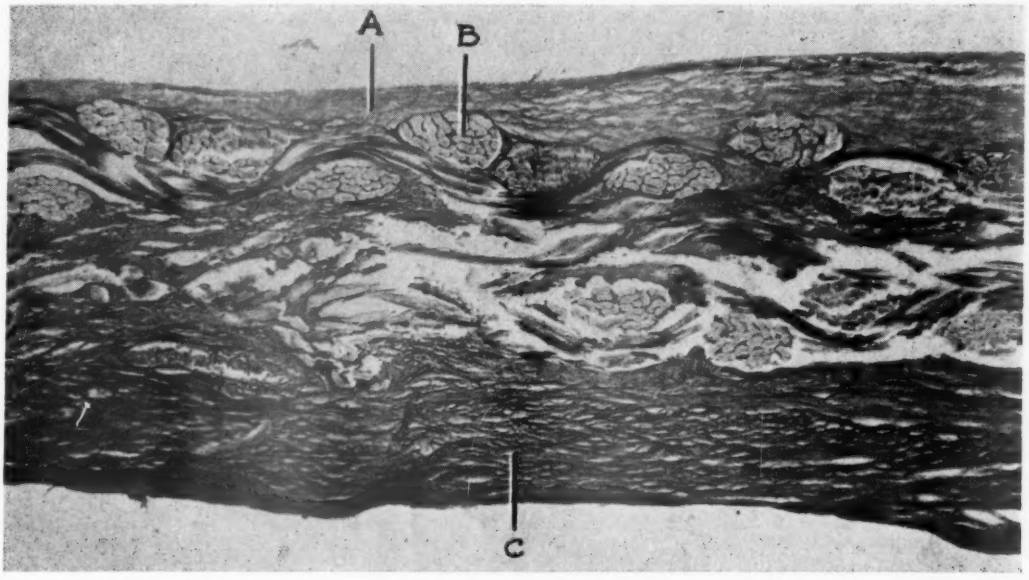


Fig. 3.—The process of arteriogenesis as shown in a longitudinal section of the newly formed aorta 73 days after the implantation of a tube of Vinyon-N mesh. *A*, A thinner layer of fibroblasts which forms the intima of the artery; *B*, a cross-section of the Vinyon-N mesh cloth serving as the elastic layer of the artery; *C*, heavy protective outer coat of fibrous tissue which forms the adventitia of the artery.

SUMMARY

In our studies,[10] which are being reported in detail, we used Orlon and Vinyon-N mesh in the form of patches and tubes. The bleeding through the meshes of the cloth of Vinyon-N, which has been mentioned by Voorhees, Shumacker, and Hufnagel, is related to the kind of weave, as well as to the physical character of the fiber itself. The bleeding has not been troublesome or serious in our experience. Orlon fiber is smoother and finer and can be woven tighter; consequently, it does not permit as much "bleeding through the mesh" and therefore has been preferred by many investigators. Both Orlon and Vinyon-N mesh are well tolerated by the body tissues and both possess a degree of elasticity which closely approximates that of the elastic layer of the aorta. Numerous attempts have been made to fashion tubes with branches or a facsimile of the aortic bifurcation. The hand-fabricated tubes make functional segments, but several investigators (Voorhees, Blakemore, Hufnagel, Shumacker, and ourselves) believe that seamless tubes of these fibers in various sizes, shapes, and with desired branches for renal arteries, common iliac arteries, and even smaller arteries, will be fabricated by some ingenious person within the near future.

The histologic studies indicate that the pervious and elastic matrix has served as a framework upon which body cells, principally fibroblasts from the

periarterial or retroperitoneal or retropleural tissues, have been able to proliferate through the meshes. This gives a smooth layer of fibroblasts which serves as the intima, with heavier coats of fibrous tissue outside the plastic mesh to serve as protective coats of the new artery (Fig. 3). Secondary contraction of the fibrous outer coat is prevented by the continued stretching due to the elasticity of the plastic mesh. We believe this is arteriogenesis and should be a practical method for replacing even long segments of large arteries, since the functional layers develop from without inwardly. This method of arteriogenesis is worthy of more serious study and further application in man.

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LONG-TERM OBSERVATIONS ON FREEZE-DRIED VASCULAR SEGMENTS AND THEIR STERILIZATION BY COBALT IRRADIATION

JAMES L. BUCHANAN, M.D., AND ALBERT G. MARRANGONI, M.D., PITTSBURGH, PA.

(From the Department of Vascular Surgery, Mercy Hospital, University of Pittsburgh School of Medicine)

THERE has been an increasing interest in the use of vascular segments preserved by the freeze-drying method or lyophilization in surgical treatment requiring bridging of arterial defects. This method of preserving vessels was first described by Marrangoni and Cecchini[1, 2] and since that time numerous investigators[3, 7] have reported satisfactory functional results experimentally. It has become popular for clinical use at the present time.[8, 10] Long-term observations on aortic homografts used in the fresh state, preserved in balanced electrolyte solution at 4° C., and those kept at –27° or –70° C. after quick freezing have been reported by Coleman, Deterling, and Parshley.[11] Because of the present popularity of the freeze-drying process, we felt that it was important to determine the long-term experience with this method as compared with those previously reported.

In the past few years high-speed electrons and radioactive isotopes have been utilized to sterilize tissue for transplantation. Meeker and Gross[12] described the use of a high-speed electron stream produced by Van de Graaff machine as a practical clinical means of sterilizing aortic grafts. MaCris and Sloan[13] reported experiments with gamma irradiation from radioactive cobalt for the same purpose. While the use of the Van de Graaff machine has apparently proved a satisfactory means of processing grafts, its usefulness is limited to relatively few centers because of the expense and technical knowledge required in procuring and maintaining such a machine.

With the widespread use of radioactive cobalt in the treatment of malignancy, it was felt that this method of sterilization would be more readily available than the previous one. Since there have been few reports on radiation sterilization of vascular segments, experiments were conducted to determine whether or not radioactive cobalt irradiation could be made a practical method for processing these grafts without altering their structure and cellular integrity.

METHODS OF STUDY

Long-Term Observations on Freeze-dried Vascular Segments.—The technique of preservation has been reported previously by one of us (A. G. M.). Femoral and aortic transplants preserved from two days to five years were implanted into twenty-five dogs by end-to-end anastomosis. No chemotherapy or anticoagulants were used postoperatively. No hindlimb paralysis or other evidence of spinal cord damage was observed as a result of occlusion of the aorta except in two dogs which developed thrombosis of their grafts at four and eight days,

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respectively. The remaining twenty-three were followed postoperatively by palpation of peripheral arterial pulses and arteriography. They were sacrificed at appropriate intervals for microscopic study of the grafted tissue.

Sterilization of Vascular Segments by Cobalt Irradiation.—Segments of aorta from pigs and adult dogs were contaminated with 4-day-old cultures of *Bacillus subtilis* and placed in sealed test tubes. The anaerobic spore-bearing organisms were chosen because they have been shown to be the most resistant to radiation sterilization.

Three methods of preparing the vessels for irradiation were used: (1) irradiation at room temperature; (2) irradiation after quick freezing at −27° C. or −70° C.; and (3) irradiation after lyophilization.

Fig. 1.—Microscopic appearance of an arterial segment which had been preserved by freeze-drying for four years.

The segments of arteries were exposed to various doses of irradiation from Cobalt^{60} ranging from 15,000r to 2,000,000r to determine the amount which would consistently produce sterile cultures. Various specimens were examined grossly and microscopically immediately after irradiation and at intervals of one, three, and six months.

RESULTS

Results on the Long-Term Observations of Freeze-dried Vascular Segments.—Vascular segments subjected to freeze-drying are essentially nonviable as determined by tissue culture. Despite the long periods of preservation, these segments took on the appearance of normal vessels after hydration with

physiologic saline solution except that they were somewhat paler. No attempt was made to determine the water content of the graft at the time of implantation. Microscopic examination of these segments following prolonged preservation showed no degenerative changes within the grafts from storage (Fig. 1).

These vascular grafts were effective as a means of bridging arterial defects. Except for the two early failures, all the segments showed open channels by arteriography. No adverse effects were noted grossly in the grafts which could be attributed to prolonged preservation. There appeared to be no more loss of elasticity of the graft wall than was present at the time of transplantation. Some increase in the fibroblastic response about the graft wall was noted after three months; however, this was indistinguishable from the adjacent normal vessel. No aneurysms developed in any of the grafts, but it was noted that there was a slight dilatation and thinning of the graft walls which could not be established as being progressive in nature. Small mural thrombi were noted in some grafts. These organized and endothelized. There were atheromatous changes noted in the 3- and 4-year-old implants. In addition, a moderate amount of calcification was found in the region of the old tributaries. There were no microscopic degenerative structural changes due to prolonged storage. A wedge-shaped thrombus formed at the site of anastomosis with some edema; and, by the eighth day, there appeared a proliferation of the fibroblasts and endothelial cells from the host. In those grafts which were stored for three years or more before being used, there was moderate necrosis of one or more layers of the graft wall following implantation. The necrosis was not extensive enough to produce weakening of the vascular channel (Fig. 2).

Degeneration of the smooth muscle was observed on the eighth day and this area became accelular and homogeneous in appearance (Fig. 3). The elastic framework appeared to maintain its normal pattern up to about one year and then there was fragmentation of the elastic tissue (Figs. 4 and 5). No definite calcification was noted within the graft wall. There was a mild fibroblastic response about the graft and this diminished in reaction from year to year. At the end of twelve to eighteen months these implants did not resemble the normal pattern except for the endothelial lining (Fig. 6). The remaining layers were composed of fibrous connective tissue with variations of the elastic framework. As previously mentioned, atheromatous changes with some calcification were noted in the intimal layer. The histologic changes found in these homografts resembled those observed with other homografts regardless of the method of preservation, the only exceptions being that there was less tissue reaction about the graft and they were more refractory to thrombosis.

Results of Sterilization by Cobalt Irradiation.—As reported by Meeker and Gross[12] and MaCris and Sloan,[13] the level of irradiation which was found to be effective in destroying spore-bearing organisms was 2,000,000 REP. Smaller doses of irradiation ranging from 50,000 REP were often sufficient to produce no growth or culture from septic segments as obtained at autopsy, but this was not constant. It was felt, therefore, that 2,000,000 REP was necessary to ensure consistent sterility. The amount of time required to obtain this level with the cobalt source used in the experiment was about two weeks. With modifications of the cobalt source, this time could be shortened considerably.

Fig. 2.

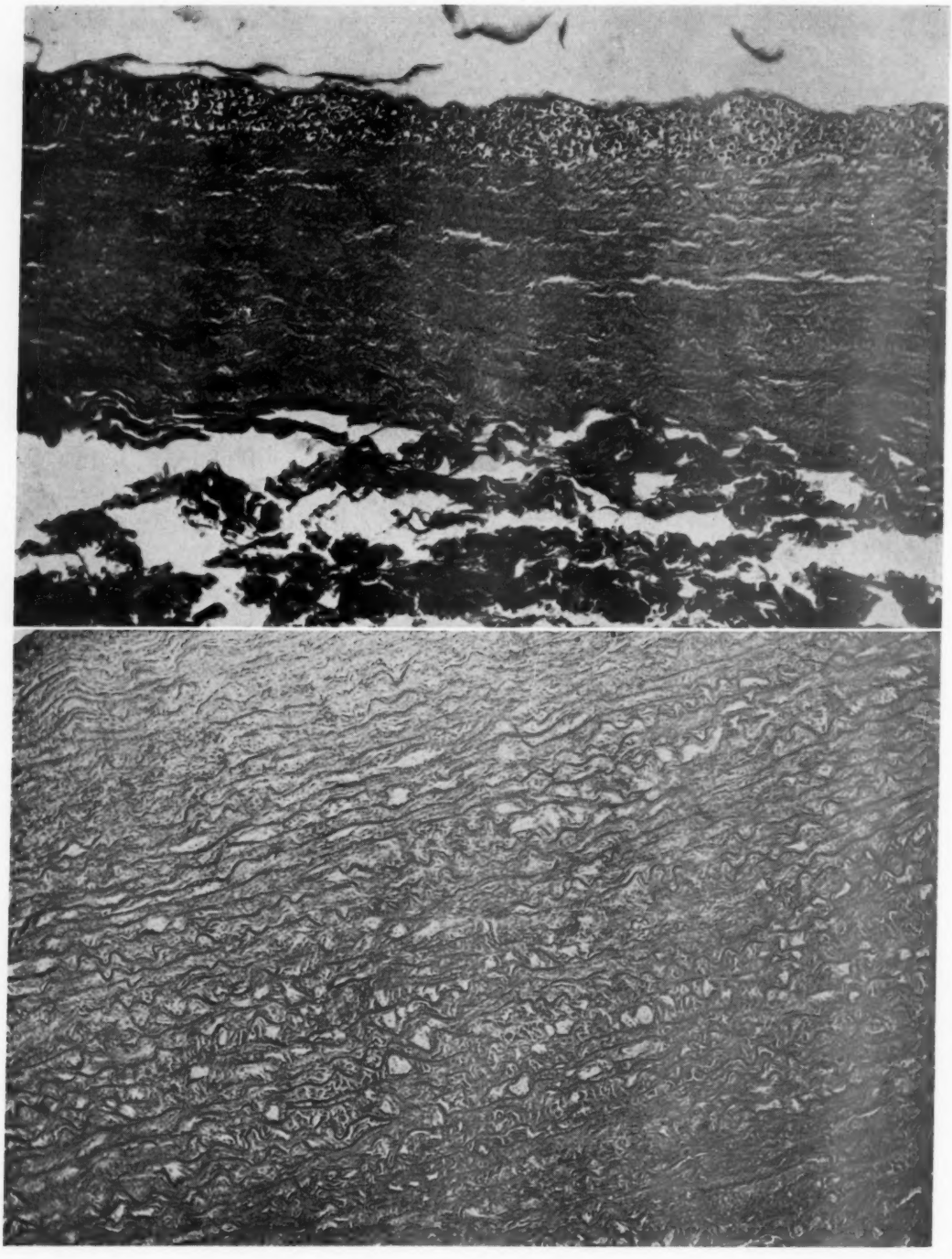

Fig. 3.

Fig. 2.—Microscopic appearance of a freeze-dried homograft in a dog ninety days after implantation. Storage time was three years and eight months.

Fig. 3.—Microscopic appearance of a freeze-dried homograft in a dog fourteen days after implantation demonstrating the loss of cellularity of the smooth muscle. Storage time was six months.

Fig. 4.

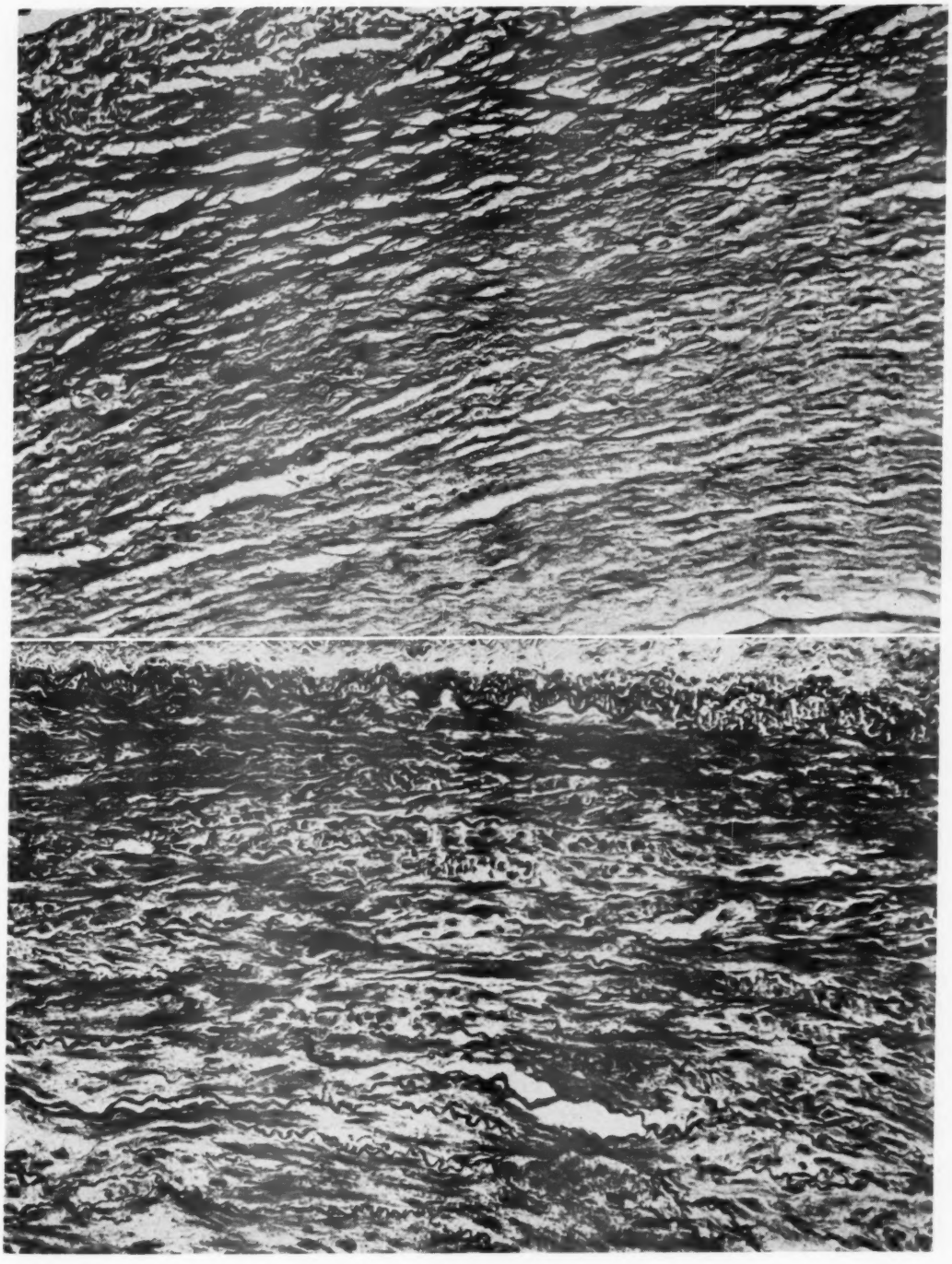

Fig. 5.

Fig. 4.—Microscopic appearance of a freeze-dried homograft in a dog one year after implantation. Storage time was eight months and three days.

Fig. 5.—Microscopic appearance of a freeze-dried homograft in a dog three years after implantation. Storage time was four months.

The segments which were irradiated at room temperature showed no growth on culture at the time of implantation but they were found to be putrified and friable. There was immediate failure when transplanted because of the severe reaction about the graft and thrombosis. Marked degenerative changes were noted in these grafts microscopically.

Fig. 6.—Microscopic appearance of a freeze-dried homograft in a dog four years after implantation. Storage time was four months.

The results of irradiation of the quick-frozen grafts were the same as with those segments which were preserved at room temperature if they were allowed to thaw while they were being irradiated. No adverse effects were noted if these segments were irradiated in their frozen state. This was found to be impractical at the present time because of the difficulty of keeping the vessels frozen for the length of time required to deliver the sterilizing dose of irradiation.

Irradiation of vessels in their freeze-dried state seemed the only practical method with the cobalt source used in these studies. The only detectable changes were in the intima (Fig. 7). For the most part, these structural changes resembled those seen in freeze-dried grafts obtained under aseptic precautions or sterilized by ethylene oxide. The fate of these grafts following implantation in dogs is being studied at the present time.

Fig. 7.—Microscopic appearance of a freeze-dried homograft exposed to sterilizing dose of cobalt irradiation (2,000,000 REP).

DISCUSSION

The long-term observations on arterial segments preserved by the freeze-drying method have demonstrated degenerative changes similar to those reported by Coleman, Deterling, and Parshley[11] and Meeker and Gross[12] with other methods of preservation. The degree of atheromatous changes observed in these grafts was not alarming and all grafts were functioning properly at the time of removal.

The strength of the graft wall does not appear to be influenced by the alterations in the elastic fibers and thinning of the graft. Although aneurysms have been reported, particularly in venous transplants, no aneurysms were noted in this series or in any lyophilized homografts which have been used clinically, to our knowledge. Because of the ease of storage, the minimal tissue reaction, and its tendency to resist thrombosis, the freeze-dried graft has become the most popular for clinical use.

Although irradiation preservation of meats, potatoes, and other food products in their fresh or frozen state has been described, the cobalt irradiation of vascular segments using this method is impractical. The length of time required for sterilization is too long as compared with other methods; however, the time interval could be shortened if the cobalt source were placed in direct contact with the graft. Irradiation of tissue at room temperature produces drastic changes in the enzyme systems and organic composition. The possibility of irradiating these vessels in their frozen state with a cobalt source in direct contact with the graft, in order to shorten the time interval required, and then allowing them to thaw and be stored at room temperature is being explored. This has not been accomplished with the ordinary cobalt unit used for irradiation therapy. It would require an additional cobalt supply with necessary shielding, making it uneconomical.

The irradiation of vessels with the cobalt source used in these experiments was found to be a satisfactory method of sterilizing contaminated vascular segments in their freeze-dried state. Freeze-drying permits their storage at room temperature for indefinite periods of time so that the time interval required for sterilization is not a crucial factor. The irradiation dosage can then be fractionated so that a cobalt source used in radiation therapy can be utilized during the intervals not required for treatment. Since there is no additional operative expense involved, this method would seem practicable for those centers which maintain cobalt units. The ultimate fate of the grafts following implantation in animals has not been studied in these particular experiments. It will be necessary to know the long-term results before recommending it for clinical use.

The sterilization of arterial segments by ethylene oxide described by Hufnagel, Rabil, and Reed[15] has proved a satisfactory means of processing contaminated grafts with little structural change. We have used this method for sterilization followed by preservation at –30° C. and have found it adequate where prolonged storage of grafts is not required. This should be useful in hospitals which do not maintain equipment for lyophilization of blood vessels. In those centers in which freeze-drying equipment is available, the sterilization by cobalt irradiation may be feasible since there is no additional expense involved and no appreciable hazards.

SUMMARY AND CONCLUSIONS

1. The long-term observations of vascular segments preserved by the freeze-drying method has been presented.

2. The histologic changes in these homografts resemble those observed with other homografts.

3. There is less tissue reaction and a minimal tendency toward thrombosis in these segments.

4. Cobalt irradiation for sterilization of blood vessels in their frozen state is impracticable with the method described.

5. The sterilization by cobalt irradiation following lyophilization was accomplished with minimal tissue destruction.

6. Further animal experimentation will be necessary before this method can be recommended for clinical use.

We wish to express our appreciation to Drs. C. R. Perryman, John Fox, Paul R. Noble, and John C. Evans of the Department of Radiology and Phillip Murray Irradiation Center of Mercy Hospital for their cooperation and technical assistance in this study.
We would also like to acknowledge the help given us by Dr. Frank X. Pawlosky in obtaining numerous arterial grafts and for his aid in our animal experimentation.

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USE OF VESSEL GRAFTS AND PLASTIC PROSTHESES FOR RELIEF OF SUPERIOR VENA CAVAL OBSTRUCTION

RALPH A. DETERLING, JR., M.D., AND SHIVAJI B. BHONSLAY, M.D.,
NEW YORK, N. Y.

(From the Department of Surgery, College of Physicians and Surgeons, Columbia University, and the Surgical Service of Presbyterian Hospital)

THE clinical syndrome of chronic obstruction of the vena cava is relatively uncommon and may escape correct diagnosis for some time. Edema of the face, neck, and arms, often intermittent, should suggest the possibility of such a diagnosis. Determination of the venous pressure in the upper extremities, standard roentgenograms of the chest, and laminography, as well as visualization of the vena cava with organic iodides, confirm the condition. Among the causes are malignant tumors of the mediastinum, especially the Hodgkin's type of lymphoma. Occasionally a large aneurysm of the ascending arch of the aorta will cause caval obstruction. When such lesions have developed sufficiently to produce obstruction, radical surgical attack is usually neither possible nor reasonable. On the other hand, one may encounter obstruction on the basis of chronic fibrosing mediastinitis or chronic tuberculosis of mediastinal lymph nodes. While obstruction of the cava initiates a very extensive collateral circulation, it never becomes sufficient to compensate completely for caval blood flow. However, in some patients symptoms are not troublesome enough to warrant surgery. Nevertheless, the vascular surgeon should be equipped with the best technique for replacement of the obstructed vena cava when indicated.

Replacement of the great veins is attended by problems of a different type from those associated with aortic grafting. The wall is thinner, more flexible, and compressible, and, although blood flow may be great, the pressures are very low. Consequently, disproportion in the diameter or length of replacement may result in angulation, buckling, or ridging of the graft, thus reducing the chances of functional success.

METHODS

Late in 1951, we began to study replacement of the superior vena cava of dogs. Since that time, twenty-four implantations have been performed. A standard technique was employed in all instances. Ventilation was controlled by a Rand type apparatus delivering oxygen and ether, following the induction of anesthesia by intravenous pentabarbital sodium (30 mg. per kilogram of body weight). The right chest was opened by a lateral intercostal incision in the fourth intercostal space. With the lung retracted, the mediastinal pleura was incised and the superior vena cava mobilized from its bifurcation to its entry into the right auricle. In dogs averaging 15 kilograms of body weight, the

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superior vena cava measured about 2.5 cm. in length. A Beck-Potts clamp usually was placed across the bifurcation of innominate veins, although in some dogs these vessels were controlled by a doubled loop of braided cotton tape. A Potts ductus clamp was placed across the vena cava at its entry into the auricle. After resection of a segment approximately 1.5 cm. in length, a replacement of similar dimensions was implanted. Two fixation sutures of 5-0 braided arterial silk swaged on a ⅜ circle taper-point needle were placed equidistant along the circumference. A posterior row of sutures was placed, using a continuous over-and-over technique, followed by an anterior row of similar type. The distal clamp was removed first in order to allow blood flow to extrude air from the graft and as a check of the suture line. The clamp adjacent to the auricle could be removed almost immediately. The total period of caval occlusion averaged twenty-five minutes.

No antibiotic drugs were instilled in the chest. The posterior parietal pleura was closed over the graft and the chest was closed in layers with silk. Full expansion of the lung was then achieved by means of a temporary intrapleural catheter and positive pressure control of respiration.

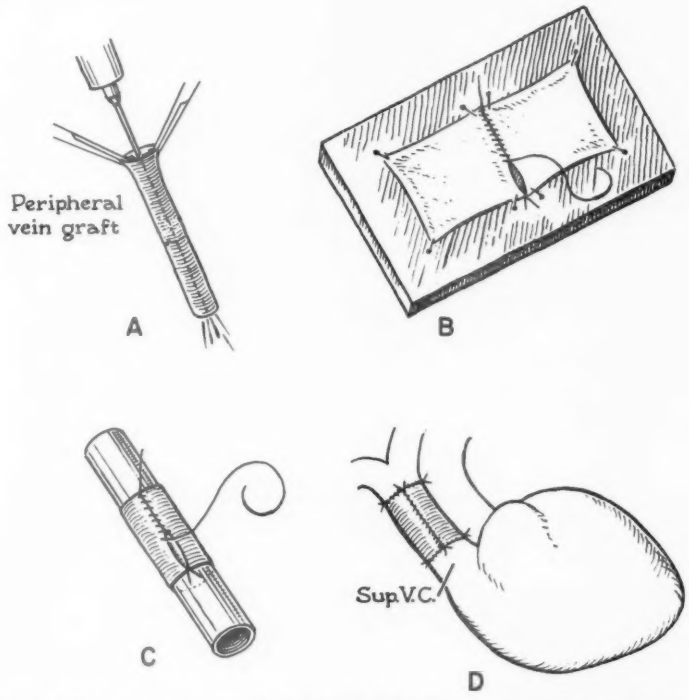


Fig. 1.—Preparation of venous autograft. *A*, Segment of peripheral vein is flushed with heparin-saline solution. The vein is then divided into two equal lengths. These are cut in the long axis to produce two panels. *B*, The panels are pinned to a cork board, and the adjacent edges sewn with 6-0 braided silk suture. *C*, The single, wide panel is then brought together over a glass tube and the free edges sewn in similar fashion. *D*, The two-panel vein graft thus prepared has a length and diameter matching the recipient superior vena cava.

A. Tissue Grafts.—

1. *Aortic homografts:* The original series consisted of four grafts of thoracic aorta stored in Simms' X6 electrolyte solution containing 10 per cent homologous serum and antibiotic drugs. These grafts had been stored at 4° C. for from one to four weeks before use. The length averaged 1.5 cm.

2. *Venous autografts:* A 4 cm. length of external jugular vein was excised and washed in a saline and heparin solution. It was then divided into two grafts af equal length. These were cut lengthwise and sewn together with 6-0 silk to produce a single vein graft 1.8 cm. long, with a diameter approximately that of the vena cava (after the method of Hurwitt and associates[1]) (Fig. 1). Four such grafts were implanted.

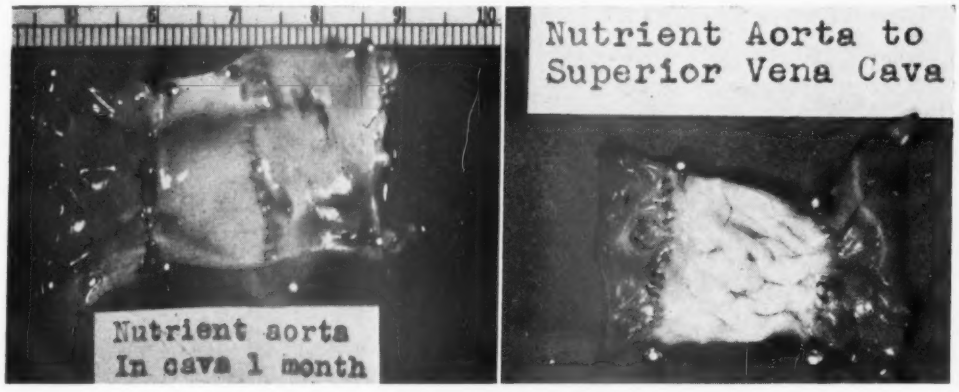
Fig. 2.—*A*, Ivalon sponge in tubular form (two sizes); *B*, cuffed tube tailored from Dacron taffeta fabric; *C*, woven seamless tube of nylon yarn.

B. Synthetic Replacement.—

1. *Ivalon tubing:* Tubing measuring 2.0 cm. outside diameter and 1.2 cm. inside diameter was supplied* (Fig. 2,*A*). Four implantations were made of lengths averaging 1.7 cm.

2. *Woven fabric:* A taffeta weave of 70 denier multifilament nylon yarn was selected because of medium porosity. The fabric was 0.0045 inches in thickness and had a thread count of 172 × 126. Tubes were fashioned by sewing the fabric with cuffed ends, using a fine needle on a standard electric sewing machine and nylon thread. A short stitch was used, with a double line of stitching at each seam (Fig. 2,*B*). This material was used in three dogs. A similar tube of a calendered† Dacron taffeta fabric was used in six animals. This cloth had low porosity and measured only 0.00375 inch thick. The thread count of 40 and 70 denier yarns was 116 × 84 after calendering, a process which gave the fabric a very smooth surface.

3. *Woven seamless tube‡:* A moderately stiff nylon tube, woven of 400 denier multifilament yarn and measuring 1.5 cm. in diameter by 1.8 cm. in length was placed in cavae of three dogs (Fig. 2,*C*).



A. *B.*

Fig. 3.—Gross appearance of aortic homografts implanted into the superior vena cava of dogs. *A,* At one month; *B,* at seven months.

RESULTS

Because of the variable length of follow-up periods of these replacements, some of them being recent, conclusions regarding long-term function cannot be drawn at this time. This report, therefore, will present the comparative behavior at time of implantation and during the first six-month period of function.

A. The aortic homografts had good elasticity and thickness of the wall so that handling and suture technique were easy. Minimal pulsation was observed when flow was resumed, however. Because of the flexibility, the anastomoses could be performed with no discrepancies in the suture line. Within a few weeks, there was an over-all contracture of the graft, associated with loss of viable cells and investment by fibroblasts of the host. The graft assumed an elliptical cross-sectional contour in the absence of distension, as with arterial pressures. After several months, the contraction process re-

*Through the courtesy of Ivano, Inc.

†A smooth surfaced texture is achieved by passing the cloth through heated rollers under pressure.

‡Supplied by Ethicon Suture Laboratories, New Brunswick, N. J.

sulted in furrows and corrigations (Fig. 3,*A* and *B*). In one graft, a 30 per cent reduction in lumen resulted from a mural thrombus, and, in all animals, the graft itself reduced in diameter as much as 40 per cent from the contraction process. Function was within satisfactory limits, with no rise in venous pressures nor edema of the head.

B. The venous autografts functioned very well initially. At the time of implantation, the graft appeared grossly and functionally identical to the adjacent cava (Fig. 4, *A*). However, complete occlusion of all four venous autografts occurred within one month. In at least two, the obstruction began at the suture line nearest the heart. At the time of obtaining cavagrams of dogs with occluded grafts, the pressure in the right external jugular vein measured from 200 to 260 mm. of saline.

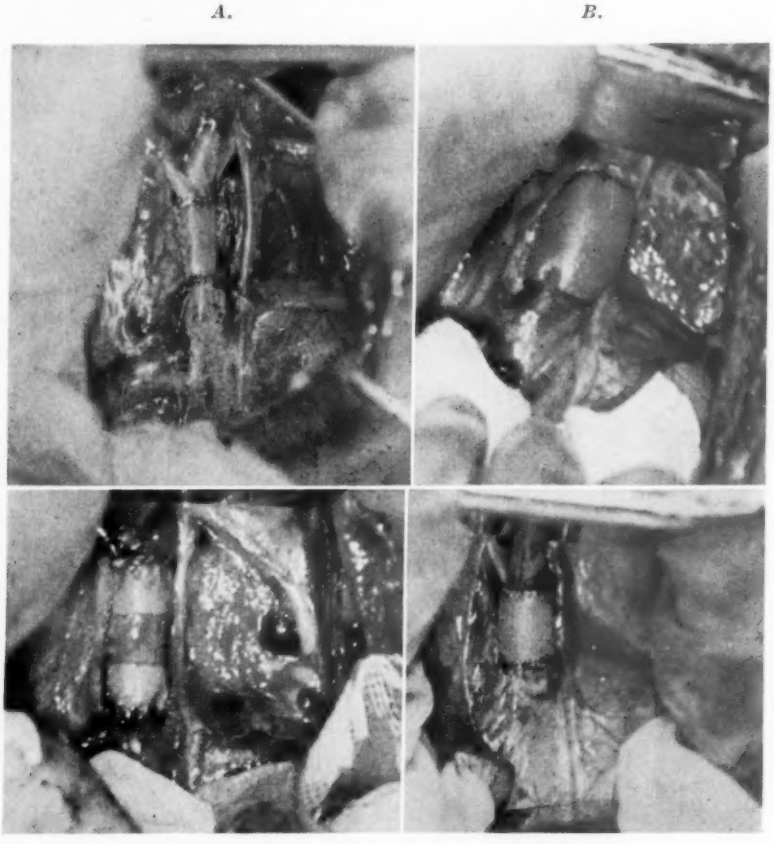


Fig. 4.—Gross appearance of functioning replacements of the superior vena cava of dogs at the time of implantation. *A*, Venous autograft; *B*, Ivalon sponge tube; *C*, tube of Dacron taffeta fabric; *D*, woven seamless nylon tube.

C. The success with synthetic materials was disappointing. The Ivalon tube was very easy to implant and the inside diameter matched that of the vena cava (Fig. 4,*B*). However, moderate reduction in lumen resulted from thrombosis, being maximal at the suture line nearest the auricle. Blood flow appeared significantly reduced as a result (Fig. 5, *B*). In none was function satisfactory.

A.

B.

C.

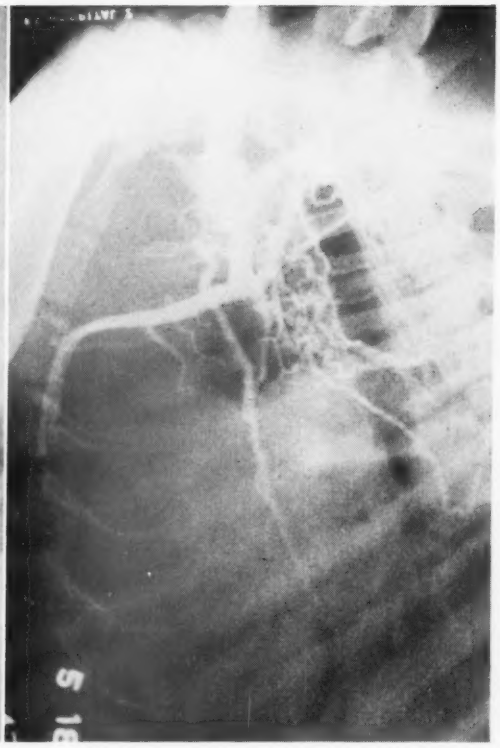


Fig. 5.—Cavagrams of dogs with replacements in the superior vena cava. *A*, Complete occlusion of venous autograft at two weeks. The excellent collateral circulation is well shown. *B*, Although slight reduction in the lumen of an Ivalon tube has occurred, there is moderate obstruction to blood flow at the lower anastomosis. Nevertheless, collateral channels are not yet evident. *C*, Complete occlusion of a Dacron fabric graft shown at seven weeks. (*For D, see following page.*)

D. Results with Dacron or nylon taffeta fabric were somewhat better. At the time of insertion of the graft, the replacement appeared satisfactory (Fig. 4, *C*). Unfortunately, because of pulmonary complications related to induction of anesthesia, empyema developed in two dogs with Dacron grafts. These thrombosed completely within a week, as was suspected by the poor clinical appearance of the animals and the presence of edema of the head (Fig. 5, *C*). In two others there was moderate reduction in the size of lumen because of thrombosis, but in the remaining two function remained satisfactory, as judged by venous pressure in the external jugular vein and by cavagrams. Among the three replacements of nylon fabric, there were two with marked reduction in blood flow as a result of thrombosis, but an excellent result was obtained in one (Fig. 5, *D*).

Fig. 5, *D*.—Graft of nylon taffeta fabric functioning well at seven weeks. Note the slight constriction at the lower suture line.

The stiff, woven, seamless tube of nylon appeared to be excellent at the time of insertion (Fig. 4, *D*) but did not pulsate with variations in blood flow. It was thought that the resistance to collapse might ensure better patency. This was not found to be so, since marked or complete thrombosis occurred within a week or two.

In summary, the results with homologous aortic grafts were superior to those with synthetic materials. Venous autografts functioned poorly, con-

trary to expectations. Better results might be achieved with the use of interrupted sutures for the anastomoses, since many failures appeared related to narrowing at the lower suture line.

Clinical Experience.—During the past four years, we have explored seven patients with obstruction of the superior vena cava. In the first of these, a tumor was present in the upper right mediastinum (Fig. 6). Exploration revealed a thymic tumor of Hodgkin's type, which had invaded the upper lobe of the lung and chest wall and invested all the great veins in this region. The patient has responded well to irradiation treatment during the past four years. In two other patients, a lymphoma was found, and irradiation was subsequently employed. In two additional patients there was a large sacculofusiform syphilitic aneurysm of the ascending arch of the aorta. Resection was not possible, and instillation of wire or use of external support was not feasible. The superior vena cava was involved in inflammatory tissue of the

Fig. 6.—Cavagram of a young woman with thymic tumor of Hodgkin's type in right upper mediastinum. There is complete occlusion of the superior vena cava and a well-developed collateral venous circulation.

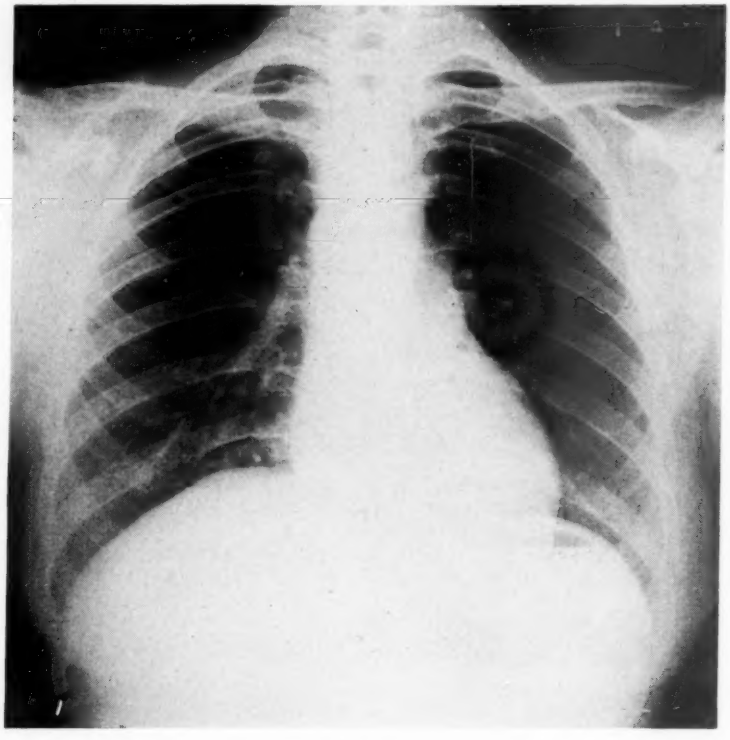
wall of the aneurysm and was flattened by expansion of the sac. These patients were alive two years later and still have evidences of caval obstruction. In one patient, not explored, there were calcific nodes in the suprahilar region of the left mediastinum. There was intermittent swelling of the upper extremities and face, but the venous pressure was only 180 mm. of saline. Cavagram revealed a partial obstruction of the superior vena cava with little evidence of collateral vessels. The patient was given antimicrobial therapy for possible tuberculosis, and has been under observation for about a year.

The remaining patient of the group subjected to operation did have complete caval and azygos obstruction from tuberculous lymph nodes. Resection of the segment and use of an aortic homograft was performed. This has functioned well during a follow-up period of eighteen months.

CASE REPORT

M. M., a 36-year-old single white woman, was first admitted to Presbyterian Hospital on Jan. 4, 1954, with a complaint of intermittent swelling of the face and arms for about one year. Past history revealed that as a child she had had an abscess under the right mandible which had to be drained surgically. It was said to be tuberculous and subsequently she received a course of irradiation to the area. She also had a long history of allergy, including hay fever and hives.

About a year before admission the patient had begun to notice an increase in the size of her arms and a filling of the facial contour. This became so noticeable that she had to alter the sleeves of her dresses and her friends frequently commented upon the difference in her face. The enlargement here was intermittent and at times was accompanied by periorbital edema. She had noted that the neck veins were more prominent and that superficial veins had become noticeable on her arms and chest. When bending



A.

Fig. 7.—*A*, Standard roentgenogram of the chest of a patient with occlusion of superior vena cava resulting from chronic mediastinal lymphadenopathy. No significant abnormalities are observed except for some calcification in the right hilar area. *B*, Laminogram at the 12 cm. level reveals a soft tissue mass with an incomplete ring of calcification in the region of the superior vena cava. The calcific area observed on the standard film is below and lateral to this. *C*, Cavagram shows a complete obstruction of the superior vena cava immediately below the junction of the innominate veins. Collateral circulation is achieved through the hemiazygos, internal mammary, transscapular and intercostal veins.

over she noted that her face became "fuller" and that her hands became red when they were in a dependent position for an extended period of time. She was extensively studied in another city where negative roentgenograms of the chest and cervical spine were obtained. She was treated for some months as a patient with angioneurotic edema. Some improvement was thought to have followed a course of cortisone. Other diagnosticians raised the question of a costoclavicular syndrome.

She had a relatively negative past history otherwise, with a stable emotional pattern. Her appetite was good. She had never had any history of cardiac, pulmonary, or renal

B.

C.

Fig. 7, *B* and *C*. (*For legend see opposite page.*)

difficulties. She had had a nontoxic adenoma removed from the right thyroid gland in 1945 and a hemorrhoidectomy in 1943. In addition she had had several courses of injections for varicose veins in 1948. Roentgenographic study of the gastrointestinal tract had previously indicated a "spastic colitis."

Physical examination revealed a slightly obese young woman in no acute distress. The skin was of normal color and texture. There were prominent superficial veins on the arms, chest, and in the neck. The arms appeared rather obese. Examination of the eyes, ears, nose, and throat was negative. There was no noticeable glandular enlargement, except that a 2.5 cm. solitary nodule could be felt in the left lobe of the thyroid near the midline. The chest was negative to percussion and oscultation. The breasts were normal. The heart was not enlarged. There were no murmurs. A_2 was greater than P_2 and there was a normal sinus rhythm. The blood pressure in the right arm was 106/70 mm. Hg and in the left arm 120/80 mm. Hg. The pulse rate was 72 per minute. The abdomen was slightly obese, but otherwise normal.

Laboratory studies revealed a hemoglobin of 12.1 Gm., red count 4.3 million cells per cubic millimeter. The white blood count was 5,500 cells per cubic millimeter with 65 per cent neutrophils, 34 per cent leukocytes, and 1 per cent monocytes. The platelet count was 251,000 per cubic millimeter. The sedimentation rate was 25 mm. per hour. The proteins of the blood were 6.2 Gm. per cent with an albumin of 4.1 Gm. per cent and a globulin of 2.1 Gm. per cent. Nonprotein nitrogen was 27 mg. per cent and the blood urea nitrogen was 9.5 mg. per cent. The urinalysis revealed the specific gravity of 1.007 and an acid reaction. Albumin and glucose were negative. There was an occasional white cell observed microscopically. A circulation time using 5 c.c. of calcium gluconate was attempted on two occasions, but no end point could be obtained. The venous pressures in the right arm averaged 275 mm. of saline. A repeat of the circulation time using 5 c.c. of Decholin was twenty-two seconds. A tracer dose of 49.7 μc of I^{131} revealed a 23 per cent uptake in twenty-four hours over the thyroid gland. The standard chest roentgenogram obtained stereoscopically in the frontal projection as well as a left lateral film failed to show any broadening of the superior mediastinum, or abnormalities of the chest wall, lungs, heart, or great vessels (Fig. 7,*A*). Laminographic studies of the superior mediastinum did show irregular calcium shadows lying just below the right upper lobe main stem bronchus, above this and somewhat medially in the angle between the upper lobe bronchus and the trachea. In the latter area there were several irregular and ring-shaped calcific contours lying 1 or 2 cm. anterior to the bronchi. The shadows were in the region of the superior vena cava and in the vicinity of the entrance of the azygos vein (Fig. 7,*B*). On an overexposed film there was the suggestion of a mass which could conceivably be obstructing the vena cava. An angiocardiogram was performed which revealed a complete obstruction of the superior vena cava just below the junction of the innominate and subclavian veins. There was an extensive pattern of collateral veins through the internal mammary and hemiazygos veins (Fig. 7,*C*). The diagnosis of complete obstruction of the superior vena cava was made with the etiologic agent presumably being chronic calcific tuberculous nodes of the mediastinum.

On Jan. 12, 1954, the right chest was entered through a posterolateral incision. The pleural space was entered through the fifth intercostal space, which permitted excellent exposure of the superior mediastinum. The pleura in the region of the superior vena cava was thickened and there was a tuberculous lymph node measuring approximately 3 by 2 by 2.5 cm. lying in the junction formed by the azygos vein and superior vena cava. This node had contacted, completely occluding both the azygos vein and the superior vena cava. The large thin-walled collateral veins noted in the visualization study were also observed. The segment of caval obstruction began about 2 cm. proximal to the heart and ended almost at the junction of the innominate veins (Fig. 8,*A*). A section of the tissue was sent for frozen sections and this examination indicated chronic inflammation, rather than malignancy. The phrenic nerve was dissected from the surface of the mediastinal pleura and pericardium and was retracted anteriorly and medially. The subclavian and innominate veins were dissected free in their bed. After cutting the posterior pleura, double loops of braided cotton tape were placed about these veins for hemostatic control. A distended, tense azygos vein was

divided and secured with a transfixion suture of 3-0 braided silk. The pericardium was entered to expose the right auricle and a 1.5 cm. segment of superior vena cava. The obstructed portion of the superior vena cava was then under control and was dissected from a very heavily fibrosed bed. Approximately 80 per cent of the node could be excised (Fig. 9,*A*). The vena caval wall was incised to determine if a contacted, patent channel were present, but this was not present. Accordingly, it was necessary to resect a 4 cm. length of vena cava and an aortic homograft of the same dimensions was implanted. A continuous suture anastomosis anteriorly and posteriorly between equidistant traction sutures of 5-0 braided arterial silk was carried out above and below without difficulty (Fig. 8,*B*). The proximal clamp was

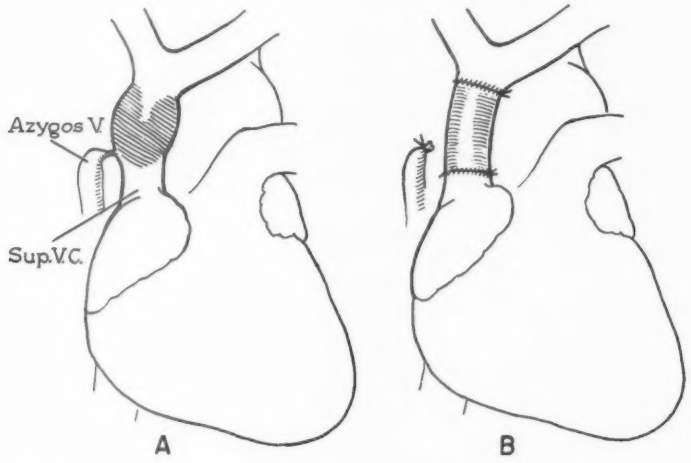


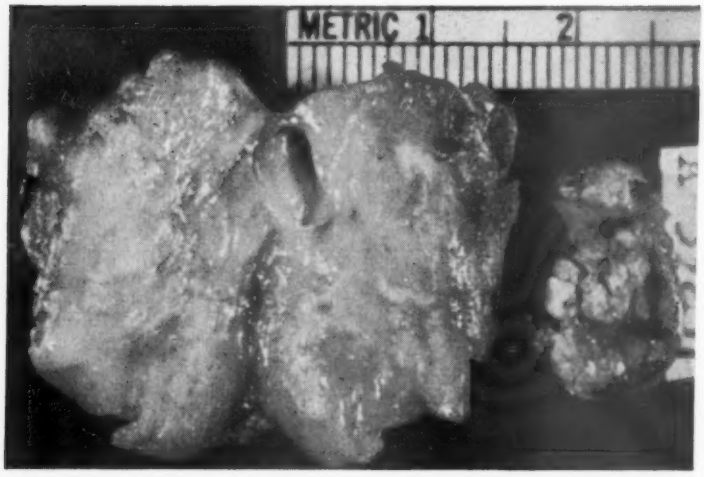
Fig. 8.—*A*, Complete obstruction of the superior vena cava and azygos vein caused by chronic inflammatory mass surrounding these structures; *B*, resection of segment of vena cava and mass was followed by insertion of an aortic homograft.

removed to allow filling of the graft and extrusion of air. There was no bleeding and the distal clamp was then removed with satisfactory restitution of blood flow through the superior vena cava. The fat pad overlying the pericardium was dissected free and swung beneath the graft to lie on the surface of the partly excised lymph node. The pericardium was only partly closed around the vena cava to avoid subsequent constriction. When the lung was fully expanded, it covered the surface of the homograft. A mixture of sulfamylon-streptomycin and 400,000 units of penicillin were introduced into the chest as the lungs were expanded. A large chest tube was placed in the eighth lateral intercostal space for drainage. The closure of the chest was carried out in layers employing silk suture technique.

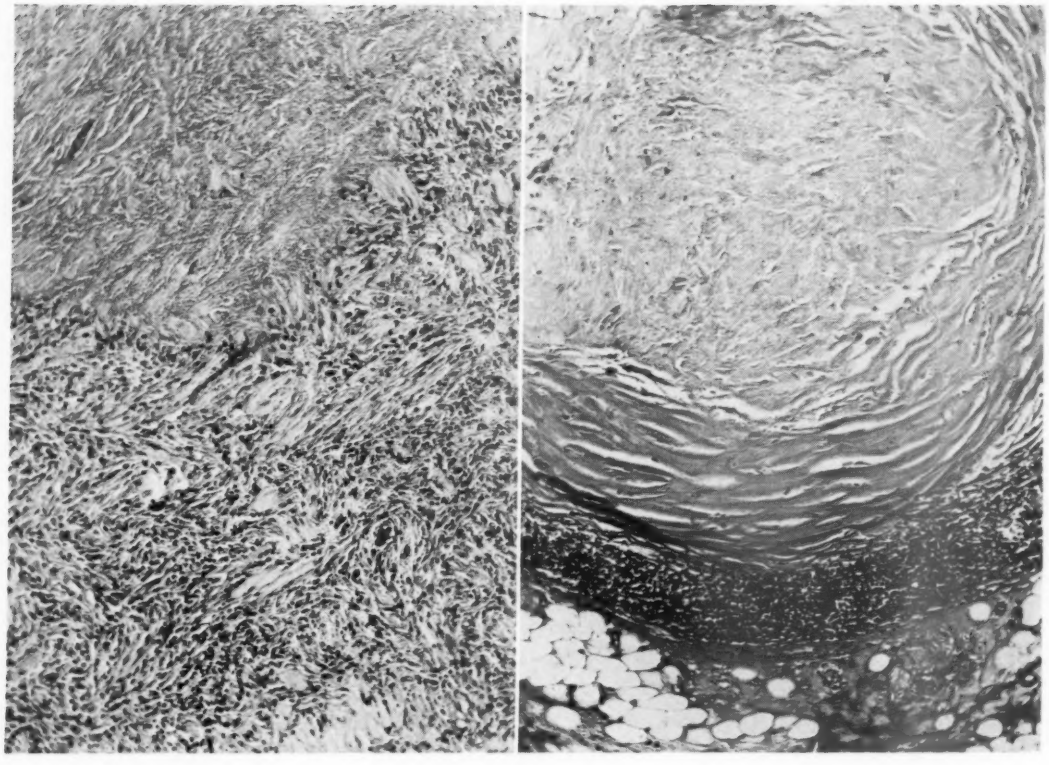
Careful examination of the removed specimen failed to reveal any acid-fast bacilli. There was a central mass of fibrous tissue with peripheral and intermingled lymphoid tissue, sometimes having well-formed lymphoid follicles (Fig. 9,*B* and *C*). No definite tubercles could be seen, yet the diagnosis was considered to be tuberculosis.

The patient had a completely uneventful convalescence. Penicillin was administered daily for 12 days in a dosage of 800,000 units daily and streptomycin was administered daily in a dose of 1 Gm. daily for six weeks. Subsequent to this, she received 1 Gm. of streptomycin twice weekly. On the eighth postoperative day, PASA (para-aminosalicylic acid) was administered by mouth in a dose of 4 Gm. daily for three days, but the oral drug was then changed to 300 mg. daily for niazide and this was continued for ten months. The patient was discharged on the fourteenth day following operation to continue on modified bed rest at home. A culture obtained from the lymph node, at the time of surgery, was negative for acid-fast bacilli.

On May 28, 1954, the patient was seen because of some coccygeal pain and numbness of the lips. The latter was considered a side effect of streptomycin dosage. An angiocardiogram



A.



B. *C.*

Fig. 9.—*A*, Calcified lymph node and inflammatory mass embracing the superior vena cava. *B*, Photomicrograph of mass reveals the margin of an area of necrosis surrounded by epithelioid cells and granulomatous tissue (hematoxylin and eosin ×150; reduced 3/7). *C*, An adjacent area reveals what appears to be an old hyalinized focus surrounded by a rim of lymphoid tissue.

obtained May 29 revealed a slightly diminished diameter of the aortic graft in the frontal projection, but adequate blood flow (Fig. 10). The venous pressures in the right arm were 140 mm. of water. A roentgenogram of the chest at this time showed minimal pleuritis at the right base. The sedimentation rate was 11 mm. per hour, a blood count revealed a hemoglobin of 14.1 Gm. and a white blood cell count of 5,000 cells per cubic millimeter, and a normal differential count. The urine revealed a specific gravity of 1.012 with negative albumin and glucose. There were 6 to 8 red cells under a high-powered field of microscopic examination. The patient was seen in the office several times during the next five months and had no swelling of the face or arms. Her general health appeared good, but she did complain of menorrhagia and dysmenorrhea. Accordingly, she was readmitted Oct. 27, 1954,

Fig. 10.—Cavagram obtained six months following insertion of aortic homograft into superior vena cava. There is apparent slight narrowing of the graft throughout its length, but no evidence of obstructed blood flow.

at which time a dilatation and curettage were performed. Microscopic examination revealed active proliferative endometrium with large, thin-walled sinuses. There was no evidence of polyps or malignancy. At this time the blood count and urine were completely normal. The venous pressure in the right arm was 52 mm. of saline. A roentgenogram of the chest on October 29 revealed little change from the previous examination. At the time of this visit the antimicrobial drugs were stopped and when last heard from, in May, 1955, her state of health was excellent and she was still employed full time as an office manager in another city.

DISCUSSION

Experiments with replacements in the vascular system date back to the last century and one of the first to transplant a free segment of vein into an artery was Gluck in 1898.[2] Within a few years, similar experiments in animals were reported by Exner,[3] Höpfner,[4] Carrel and Guthrie,[5] Carrel,[6] Watts,[7] Stich

and associates,[8] and others. Variable and sometimes discouraging results attended these efforts in the period before antibiotic drugs and improvements in anesthesia and blood replacement. More recently, better results were recorded by Blakemore and Lord[9] and Johnson and co-workers.[10] These authors also used free vein grafts in peripheral arteries of human beings with success, as have others.[11-13] Late aneurysm formation was observed when caval autografts were transplanted into the aorta of dogs, however.[14, 15] Autologous vein segments have been used as inlay grafts in arterial and aortic aneurysms, but with limited success.[16, 17] The use of homologous caval transplants in the aortas of dogs failed to show any advantage over aortic homografts.[18]

Less information is available regarding transplantation of veins into the venous system since most veins may be sacrificed without significant sequelae. However, free vein autografts have been employed successfully in the treatment of portal hypertension. Segments of superficial femoral vein have been used to bridge the gap between splenic vein and renal vein, and between portal vein or tributary and inferior vena cava.[19-21] Limited success with tissue grafts, synthetic fabric, and plastic tubes in the external jugular vein, portal vein, inferior vena cava, and superior mesenteric vein of dogs has been reported by Egdahl and associates[22] and Daniel.[23] They observed better results with tissue grafts than with plastic tubes. Orlon fabric replacements were unsatisfactory.

The use of replacements for obstructive lesions of the vena cava has been infrequent. A review of the subject in reference to lesions involving the superior vena cava was made by Klassen, Andrews, and Curtis[24] in 1951. In one patient these authors used a segment of peripheral vein implanted between azygos vein and auricle, employing an 8 mm. Blakemore-Lord Vitallium tube. There was partial decompression of the obstructive lesion during the short period of follow-up. In 1954, Scannell and Shaw[25] reported two patients in whom free segments of superficial femoral vein were used to bridge an obstructed segment of superior vena cava. In the first patient the graft extended from the right jugular vein to the right atrium. It was necessary to ligate the right subclavian as well as the left jugular and subclavian veins. The patient died on the eighth postoperative day, but despite the presence of an acute mediastinitis, the vein graft was patent. In the second patient, the graft was implanted in similar fashion. Although improvement was noted subjectively, the brachial venous pressure was 23.6 cm. of saline three months following operation. This was of doubtful significance since the subclavian artery had been ligated. However, there was still congestion and reddening of the face if the patient bent forward while seated. A venogram revealed some constriction at the upper anastomosis.

Samson[26] reported partial success in a patient with chronic mediastinal lymphadenopathy who had been irradiated because of a presumed diagnosis of lymphoma. A free graft of superficial femoral vein was used end-to-end at the upper end and end-to-side at the level of the atrium. Although symptoms had improved, the venous pressures remained elevated. Kay[27] had

tried caval homografts in dogs with only 30 per cent success and subsequently he operated upon three patients with caval obstruction secondary to chronic fibrosing mediastinitis. In the first, a venous homograft was implanted between the innominate vein and auricular appendage, but the patient died in the early postoperative period. In the second case, the azygos vein was anastomosed to the auricular appendage without lasting success. At four months, the anastomosis had closed. In the third case, an autograft of superficial femoral vein was used between the innominate vein and auricular appendage. During the follow-up period of five years, the graft ceased functioning. Hanlon[28] reported the use of an aortic homograft to by-pass a caval obstruction from malignant tumor. The graft was inserted into the end of the auricular appendage and into the side of the vena cava above the obstruction. The graft appeared to function for the remaining four months of the patient's life.

In 1954, Holman[29] reported a patient with inflammatory obstruction of the superior vena cava in whom a segment of preserved aorta was used. The graft was implanted in the defect with end-to-end anastomoses but subsequent follow-up indicated that the graft had become at least partially occluded at about twelve months.

In 1955, Blum, Medl, and Keefer[30] reported the use of an aortic bifurcation homograft for a congenital absence of the subrenal portion of the vena cava. The patient had extensive collateral veins of the abdominal wall with chronic ulceration in this region. In a short follow-up period the graft appeared to be functioning well. DeBakey[32] has also used freeze-dried aortic bifurcation homografts for replacement of both the terminal aorta and inferior vena cava in a young man with a leiomyosarcoma. The function was apparently satisfactory several months later.

It is of interest that the reconstruction of the superior vena cava in the reported cases, as in ours, was for obstruction produced by a chronic inflammatory process, in all but one case reported by Hanlon (Table I). In one case reported by Scannell and Shaw, irradiation had been given for a tumor but at operation no evidence of a neoplastic process was found. In the case presented by Holman a mass had been noted, but this was found to be a mass of lymph nodes at time of exploration.

Experimental work on the problem of caval replacement has been sparse despite the relatively high incidence of unsuccessful results clinically. Gerbode, Yee, and Rundle[31] in 1949 reported the anastomosis of superior vena cava to auricular appendage after transection. Although the results were encouraging, several failures were observed with this technique. The work with caval homografts by Kay has been mentioned. His success in only 30 per cent of the dogs corresponds to our trial with synthetic materials. DeBakey[32] has also observed only about 25 per cent success with aortic homografts in the venae cavae of dogs.

The partial success in clinical cases with femoral vein grafts suggests that special attention should be given to the diameter of the transplant and to the

suture technique. Some of the partial failures in the clinical cases were related to thrombosis or constriction at the anastomosis.[24, 26] This was observed in our experimental failures as well. It is our belief that the use of an interrupted anastomosis may be of benefit. We have not tried catgut suture material, although we have found it satisfactory in arterial vascular anastomoses.[33]

The use of an auricular appendage would seem theoretically less desirable than the cava for a line of anastomosis, and where this is impossible an end-to-side anastomosis to the wall of the atrium might be preferable. The appendage varies in size, has trabeculations, and there is considerable free motion. It is noteworthy that all but one of the autologous veins implanted into auricle or appendage of clinical cases had either partial success or failed. A single case had a patent vein graft but the patient died only eight days following implantation (Table I).

TABLE I. SUPERIOR VENA CAVAL OBSTRUCTION TREATED BY REPLACEMENT

SURGEON	CASE	ETIOLOGY	TYPE OF GRAFT	TYPE AND SITE OF ANASTOMOSIS	RESULT
Klassen[24]	1	Inflammation	Femoral autograft	Vitallium tube: azygos-atrium	Partial success
Scannell and Shaw[25]	2	Inflammation (irradiated)	Femoral autograft	Suture: rt. jugular to atrium	Died 8 days; graft open
	3	Inflammation	Femoral autograft	Suture: rt. jugular to atrium	Partial success
Samson[26]	4	Inflammation (irradiated)	Femoral autograft	Suture: vena cava to atrium	Partial success
Kay[27]	5	Inflammation	Femoral autograft	Suture: innominate to appendage	Failure
	6	Inflammation	Caval homograft	Suture: innominate to appendage	Died early postop.
Hanlon[28]	7	Malignant tumor	Aortic homograft	Suture: side of cava to appendage	Graft open (died at 4 mo.)
Holman[29]	8	Inflammation	Aortic homograft	Suture: cava to cava	Failure suspected
Deterling	9	Inflammation	Aortic homograft	Suture: cava to cava	Graft open at 18 months

In conclusion, our review of experimental studies and nine human beings with replacements of the superior vena cava indicates that partial or complete failure of the graft is the most frequent result. Our trial of several types of replacement was discouraging, although we did have encouraging results with aortic homografts. We plan to continue our studies, with major emphasis on the anastomosis. It may be that greater success could be achieved experimentally if these grafts were placed in an obstructed cava under higher pressure, as is present clinically. However, with the fall in pressure subsequent to relief of obstruction, this advantage would be nullified.

Antibiotic therapy should be employed with the use of grafts, since infection, at least in the very early postoperative period, probably contributes to thrombotic occlusion. If tuberculosis is suspected, we believe a course of specific drugs should be administered before and following operation.

SUMMARY

An experimental study has been presented in which various types of replacement of the superior vena cava of dogs were evaluated. Although some success was observed with aortic homografts, the results with materials such as Ivalon sponge, taffeta of Dacron and nylon, and woven nylon tubing were discouraging. Venous autografts also failed even though equivalent diameter was achieved with peripheral veins, by use of the panel method of making the graft. Interruption of the suture line may be important, since most failures occurred at the anastomosis. The relatively poor clinical experience in eight collected cases indicates the need for further investigative work on the subject of replacement for chronic obstruction of the superior vena cava. We have presented a case in which an aortic homograft has functioned successfully for eighteen months.

We wish to acknowledge the participation of Drs. Joseph Jailer and William Stearns in the diagnostic and therapeutic management of the patient described in this paper. We express our thanks to Dr. Vance Lucas for his help in the implantation of aortic homografts in dogs, to Dr. Raffaele Lattes for his help in interpreting the pathologic material from the clinical case, and to Miss Daisy Mapes for her assistance in the experimental laboratory. The suture material used in the experiments was donated by J. A. Deknatel and Son.

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THE AFFERENT LOOP SYNDROME

GEORGE L. JORDAN, JR., M.D., HOUSTON, TEX.

(From the Department of Surgery, Baylor University College of Medicine, and the Surgical Service, Veterans Administration Hospital)

FOLLOW-UP studies of patients treated for complications of gastroduodenal ulceration by subtotal gastrectomy reveal that approximately 85 to 90 per cent of them are improved. Some patients classified as having poor results are relieved of their preoperative disease only to develop new postprandial disturbances in the postoperative period. Several years ago it was common practice to refer to all such symptoms as "dumping" or the "postgastrectomy syndrome." However, recent investigations have demonstrated that symptoms may arise from a multiplicity of causes and proper management depends upon an accurate evaluation of the underlying pathophysiology.[3, 7, 12, 14] One syndrome which has received considerable comment in the English literature but which still is not recognized by many American surgeons has been referred to as the "afferent loop syndrome." In 1942, McNealy[6] discussed the problem of afferent loop obstruction in the early postoperative period and stated that it was a significant factor in the problem of leakage from the duodenal stump. However, he did not discuss in any detail the difficulties which may arise in the late postoperative period. Lake[4] was one of the first to recognize the possibility of chronic partial afferent loop obstruction as being the cause of symptoms in the late postgastrectomy period but felt that it was a rare phenomenon. Muir[8] in 1949 and Steinberg[11] in 1950 reported cases of afferent loop stasis which they had treated surgically. However, despite these earlier comments on the subject, Wells and Welbourn[14] in 1951 were the first to discuss in detail the etiology, symptomatology, and treatment of this disorder. Whereas many articles have appeared in the English literature which deal with this subject, it seems to have been almost completely ignored in the American literature despite a large number of published reports concerned with investigation of postgastrectomy symptoms.

PATHOGENESIS

This condition develops only after the Billroth II type of gastrectomy, as symptoms are due to partial or recurrent obstruction of the afferent loop prohibiting the passage of bile and pancreatic juices from the duodenum into the efferent loop. Obstruction may be due to adhesions, to the weight of a distended colon, to an internal herniation, or to volvulus. Wells and MacPhee[13] stated that it is most likely to be seen following the Finsterer-Lake type of antecolic right-to-left anastomosis associated with a Hoffmeister valve, when the afferent loop is long, or when a very high resection has been performed, as acute kinking of the bowel occurs more readily under these circumstances. This

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syndrome differs from the acute obstruction of the afferent loop due to herniation under the efferent loop.[10, 15] Acute obstructions are usually complete and progressive until there is surgical intervention. The vomitus contains no bile. In the "afferent loop syndrome" the pathophysiology gives rise to chronic recurring symptoms rather than one acute episode and the vomitus does contain bile, as vomiting usually occurs when the contents of the afferent loop are emptied.

The degree of obstruction may be mild or severe, but it is not complete for any long period. Symptoms are produced during the period of obstruction and are relieved when the contents of the afferent loop are evacuated.

SYMPTOMS

In the typical case, the patient complains of nausea occurring ten to twenty minutes after eating and persisting for from a few minutes to one hour. Vomiting may occur after each meal or only after an occasional meal. When vomiting occurs the patient is immediately relieved, and symptoms do not recur until more food is ingested. The character of the vomitus is significant in that it usually consists of bile-stained fluid unmixed with food, and in volumes as great as 500 to 1,000 ml. If vomiting is severe, food is finally ejected as the disturbed motility spreads further down the gastrointestinal tract, but in such cases bilious regurgitation always precedes the appearance of food particles.

The accumulation of bile and pancreatic juices in the partially obstructed loop results in distention which, in association with the increased motility necessary to overcome the obstruction, may result in a feeling of fullness, sometimes amounting to pain. Vomiting occurs only when the pressure in the afferent loop rises sufficiently high to propel suddenly the contents of the duodenum into the stomach and efferent loop; consequently, the duodenum may remain distended for several hours, or, in severe cases, several days. Under these circumstances there is a feeling of fullness, nausea, and anorexia made worse after ingestion of food, and these symptoms are finally relieved when vomiting occurs. Symptoms are most severe after a fatty meal which results in a maximum liberation of bile.

The symptoms presented by these patients resemble those seen in other postgastrectomy syndromes; however, weakness, sweating, and pulse rise are less common. Symptoms may be minimized if the patient is recumbent in the immediate postprandial period as this relieves the downward pull of the full stomach and therefore may decrease the degree of obstruction.

DIAGNOSIS

Undoubtedly one of the reasons why this disorder has not been separated from others is that there have been no objective tests to aid in evaluating the patient's complaints. In most reported cases the diagnosis has been made on symptomatology alone.

Wells and MacPhee[13] have noted that barium rarely passes into the afferent loop during a gastrointestinal series when obstruction exists, although failure of

barium to enter the afferent loop does not necessarily mean that obstruction is present, as normal peristalsis may carry all of the barium into the efferent loop. Lorber and Shay[5] have reported that filling of the afferent loop can be accomplished when desired if the efferent loop is obstructed with a balloon in order to gain pressure. In this manner they have demonstrated several types of pathology which could not have been demonstrated by conventional techniques; however, they do not report the use of this maneuver for demonstration of afferent loop obstruction. In our experience, roentgenography has been a useful diagnostic procedure. Whereas little or no filling of the afferent loop may be seen following the ingestion of small amounts of barium, this segment of bowel will almost invariably be outlined, without the use of special maneuvers, if the patient ingests barium until he feels uncomfortably full. If this procedure fails, the technique of Lorber and Shay may then be utilized. Failure to fill the proximal bowel with this method is definite evidence of obstruction to the retrograde flow of barium, and has the same connotation as a similar finding in other parts of the gastrointestinal tract.

When the roentgenographic findings are definite, little doubt of the diagnosis exists; however, since symptoms may occur intermittently, it is possible that a gastrointestinal series may be performed at a time when obstruction is minimal, and the diagnosis might be missed. In an effort to gain additional objective evidence to aid in evaluation of patients with suspected afferent loop obstruction, a test was devised, predicated upon the fact that normally one finds varying amounts of bile in jejunal contents at all times. A mercury-weighted, large-lumen Levine tube is passed into the efferent loop 6 in. to 12 in. beyond the gastrojejunostomy stoma, and its position confirmed by a roentgenogram of the abdomen. A fasting specimen is aspirated, and the patient is then fed a liquid meal consisting of 60 ml. of Ediol,* 240 ml. of milk, and 180 ml. of 40 per cent glucose solution. Aspirations of samples of the jejunal content for inspection are made at one-minute and then at five-minute intervals until good mixing of bile with the ingested meal is seen. If afferent loop obstruction is present, there should be an absence of bile during the period of symptoms, and relief of symptoms should occur when bile passes the obstructed point.

The results of this test in 10 control postgastrectomy patients revealed that the ingested meal could often be aspirated within one minute after the first swallow. Bile mixing is grossly discernible within the first five minutes, and complete mixing occurs within ten to twenty minutes. In patients with other postgastrectomy difficulties, there is no correlation between bile mixing and the presence of symptoms. A positive test, which indicates the presence of afferent loop obstruction, is recorded when there is no discernible bile present in the jejunum during the period of symptoms, and the appearance of bile coincides with the disappearance of symptoms. Our experience with this test in patients in whom afferent loop obstruction was suspected clinically is limited; however, the results have been so strikingly confirmatory of the

*Ediol is an oral fat emulsion prepared by Schenley Laboratories, Inc., the main constituent of which is coconut oil 50 per cent.

suspected pathophysiology that we feel its use will be of considerable diagnostic aid in future cases. The results of this test are shown in Fig. 1. The upper column shows the jejunal content in the fasting one-, five-, ten-, thirty-, and sixty-minute specimens aspirated from Patient H. W. D. Comparison with the appearance of the ingested meal shown to the far right of the figure demonstrates that good bile mixing did not occur for one hour following ingestion of the meal. Jejunal contents aspirated from an asymptomatic postgastrectomy patient are shown in the lower column. Varying degrees of bile mixed with the ingested meal may be seen in all samples.

The test herein described is of particular value in that it can be performed during a period when the patient is actually experiencing symptoms so that one is able to obtain a positive correlation between objective findings and subjective symptoms.

TREATMENT

The treatment is surgical. Symptoms may be relieved by any procedure which eliminates the site of obstruction. A jejunoplasty as described in detail by Steinberg[11] provides a satisfactory reconstruction if the obstruction is due to kinking at the anastomosis. Steinberg has performed this procedure on 25 patients for postgastrectomy symptoms and has reported complete relief in all but 2 instances. Wells and Welbourn[14] have utilized it in 14 patients, also with good results. Muir[8] has diverted the afferent loop secretions from the stoma by making an anastomosis between the afferent and efferent loops. Schofield and Anderson[9] have suggested a Roux-Y type of anastomosis which would prevent afferent loop stasis. The latter 2 procedures undoubtedly will relieve afferent loop obstruction; however, most observers concede that such procedures are likely to increase the incidence of marginal ulceration despite Schofield's statement that none of the 90 patients treated in this manner has developed this complication.

Two patients have been seen in our Gastrointestinal Follow-up Clinic who presented the clinical picture of a chronic afferent loop obstruction and have been successfully treated by operation.

CASE REPORTS

CASE 1.—E. B., a 57-year-old white man, was first seen in February, 1952, at which time he gave a long history of duodenal ulcer, complicated on this admission by an acute perforation. A subtotal gastrectomy was performed and gastrointestinal continuity reestablished with an antecolic Hoffmeister gastrojejunostomy 6 inches distal to the ligament of Treitz. He was readmitted in November, 1953, stating that he had remained asymptomatic after operation until October, 1952, at which time he developed postprandial substernal and right upper quadrant pain with associated nausea and vomiting. Diagnostic studies failed to reveal any significant abnormality and the patient was discharged on an ulcer regimen.

He was readmitted in February, 1954, stating that his symptoms had continued without relief. He no longer had substernal pain, but a dull right upper quadrant pain persisted. His main symptoms were those of nausea and vomiting, which occurred after almost every meal. Symptoms would appear twenty to sixty minutes after eating and would usually persist until he vomited, following which time he would remain asymptomatic until he ate again. The vomitus never contained recently ingested food, but consisted only of bile-stained fluid. While in the hospital he at times vomited as much as 1,000 ml. of this fluid.

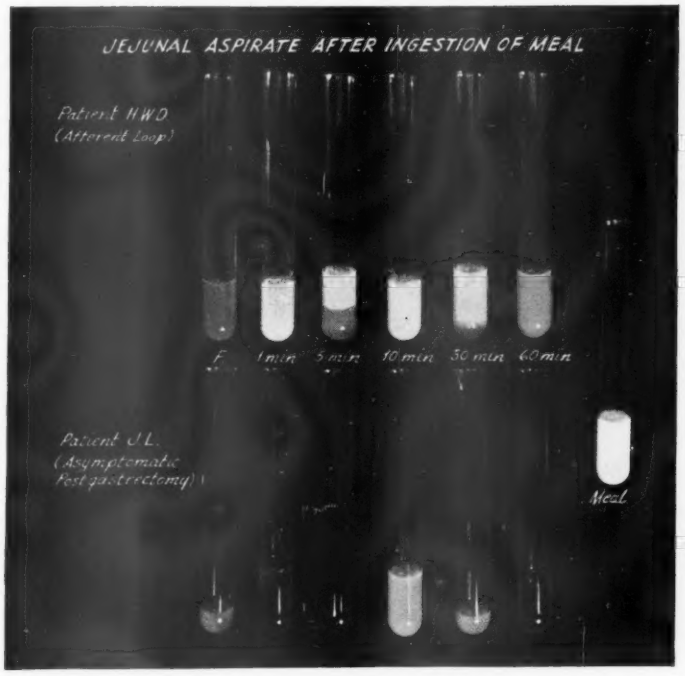

Fig. 1.—Comparison of the appearance of jejunal contents following ingestion of liquid meal aspirated from patient with afferent loop obstruction with that from an asymptomatic postgastrectomy patient.

There were slight abdominal distention and mild tenderness in the right upper quadrant. Laboratory examinations were as follows: Hemoglobin 11 Gm. per 100 c.c., serum bilirubin 2.0 mg. per cent and Bromsulphalein retention of 30 per cent at thirty minutes. Other liver function tests were within normal limits. A gastric analysis showed no free acid. A roentgenogram of the gall bladder showed only faint visualization, but no stones were seen, and a gastrointestinal series was reported within normal limits. However, a special barium study with a balloon occluding the efferent loop showed obstruction to the retrograde flow of barium into the afferent loop. On the basis of the clinical picture and these laboratory studies, diagnoses of chronic afferent loop obstruction and of cirrhosis of the liver were made. On March 3, 1954, an exploration revealed that adhesions had formed between the afferent loop, the liver, and the lesser curvature closure of the stomach with acute angulations which would not permit passage of a No. 14 Fr. catheter. The adhesions were at a point where filling of the stomach would increase the degree of obstruction

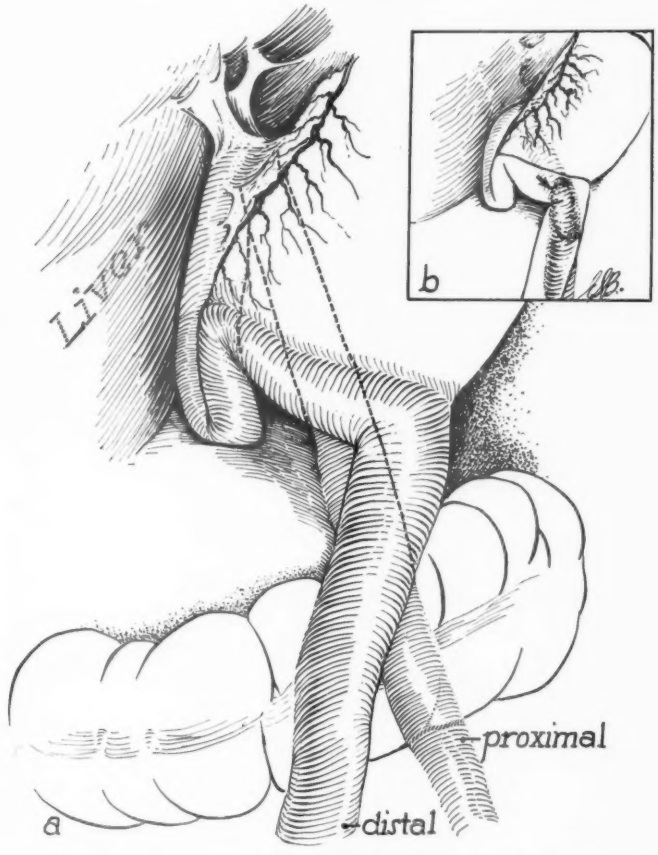

Fig. 2.—*a*, Obstruction of afferent loop resulting from adhesions to stomach and liver; *b*, obstruction relieved by lysis of adhesions and enteroenterostomy.

(Fig. 2, *a*). The diagnosis of cirrhosis was confirmed grossly and microscopically. The gall bladder, the extrahepatic biliary tree, and the pancreas were normal to palpation. The adhesions were lysed and an enteroenterostomy was performed between the afferent and the efferent loops at the level of the gastrojejunostomy stoma, so that the obstruction was released without diverting the bile and pancreatic juices away from the anastomotic site (Fig. 2, *b*). He became asymptomatic in the immediate postoperative period and when last seen twelve months postoperatively he had remained symptom-free. Liver function tests at this time were all normal except for 10 per cent retention of Bromsulphalein at thirty minutes.

CASE 2.—H. W. D., a 26-year-old white man with a two-year history of peptic ulcer, underwent a subtotal gastrectomy with an antecolic Hoffmeister gastrojejunostomy on Sept. 10, 1952. He remained asymptomatic until December, 1953, at which time he developed nausea and vomiting after almost every meal. The vomitus consisted of bile-stained fluid without food, and at times as much as 1,000 ml. would be regurgitated. Physical examination was completely negative and all laboratory procedures were within normal limits except for the test described previously. (Fig. 1). Fasting aspirate from the jejunum revealed only a slight trace of bile. After ingestion of the liquid meal no bile could be seen mixed with the meal for thirty minutes, and complete mixing did not occur for one hour. During the period when the bile was absent from the jejunum the patient was quite nauseated.

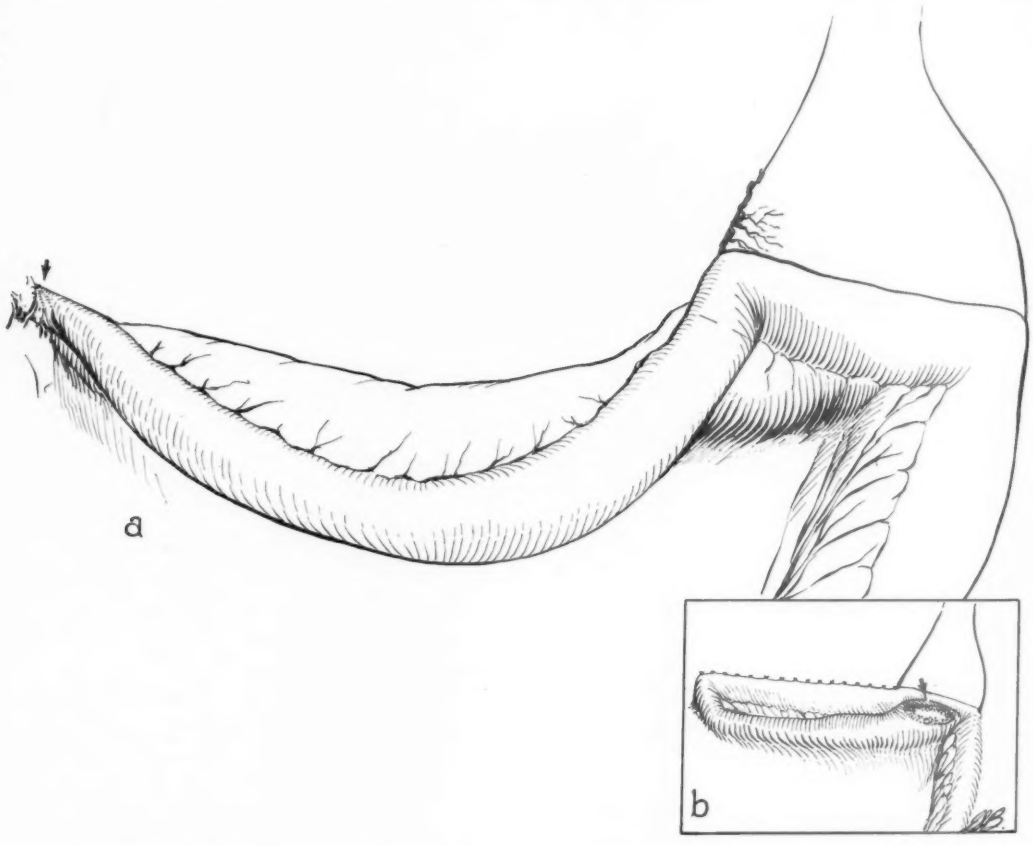

Fig. 3.—*a*, Obstruction of afferent loop resulting from volvulus; *b*, obstruction relieved by suturing bowel to prevent volvulus and by an enteroenterostomy which, in effect, creates a short afferent loop.

He did not vomit, but his symptoms disappeared when good mixing had occurred. Operation was performed on April 20, 1954, at which time it was found that a long afferent loop had been used due to a congenital retroperitoneal displacement of the first part of the jejunum so that the jejunum became intraperitoneal in the right upper quadrant (Fig. 3, *a*). A volvulus occurred around this fixed point, which was influenced by changes in position and by changes in volume of the stomach. The loop was fixed in place by sutures so that volvulus could not recur, and an enteroenterostomy was performed which, in effect, created a short afferent loop (Fig. 3, *b*). The postoperative period was uneventful and within two months he had gained 20 pounds. When last seen ten months postoperatively, he was asymptomatic.

DISCUSSION

The vomiting of bile unmixed with food has been noted by many observers. The most common explanation has been that it was a feature of the "dumping syndrome" in which food passed quickly into the efferent loop, caused nausea, and when vomiting occurred the only material in the region of the stomach was bile-stained fluid. Boman[1] has observed that the gall bladder may be evacuated with a very forceful contraction after the stomach is empty, and he believed that it was this rapid emptying of the gall bladder which caused such symptoms. It is quite possible that both of these factors could result in the vomiting of small amounts of bile-stained fluid without food, but it is unlikely that as much as 500 or 1,000 ml. would be vomited under such circumstances.

The fact that these symptoms may mimic those found in other syndromes makes it necessary to have objective confirmation of the diagnosis, for most patients with other types of postprandial distress following gastrectomy are satisfactorily managed by nonoperative means, whereas surgical intervention is the procedure of choice when a mechanical obstruction to a portion of bowel exists. The test of bile aspiration is a simple one which can be accomplished as readily as a gastric analysis. In fact, aspiration of stomach contents might well give the same information, although jejunal aspiration seems preferable, since bile will always enter this segment of bowel unless it is vomited, whereas it is conceivable that it might by-pass the stomach to a large extent. The passage of a tube into the jejunum is accomplished quite easily in a patient who has had a gastric resection. No special maneuvers are needed, and in some patients it passes almost immediately. The technique commonly used consists of passing a mercury-weighted tube into the stomach with sufficient slack and allowing the patient to be ambulatory for one hour, at the end of which time a roentgenogram of the abdomen is made. I have passed jejunal tubes in many postgastrectomy patients for this test and for studies of intestinal motility, and failure of passage spontaneously within one hour is very rare.

The finding of biliary cirrhosis in the first patient and the presence of mild jaundice is interesting. Although Wells and MacPhee[13] mentioned the fact that occasionally mild icterus may be noted, he reported no cases in which definite liver damage was observed. It is impossible to state absolutely whether the cirrhosis and jaundice resulted from chronic afferent loop stasis in this man. However, it is conceivable that recurrent or chronic obstruction causing an increased pressure in the afferent loop might impede the flow of bile from the liver and therefore have an effect similar to that of a partially obstructing common duct stone.

Periodically, reports are published concerning the use of a specific surgical procedure for alleviation of the "dumping syndrome."[2, 11] As pointed out by Wells and MacPhee,[13] it is likely that many patients who have obtained relief from surgical procedures have actually had an unrecognized afferent loop

obstruction. In fact, Wells and Welbourn[14] believed that a component of afferent loop obstruction is present in many patients who also have symptoms on another basis. Schofield and Anderson[9] stated that most postgastrectomy symptoms are due to afferent loop stasis. The experience in this clinic does not support these contentions, for we have found no statistically significant difference in the incidence of postprandial symptoms occurring after the Billroth II as compared with the Billroth I procedure, and since patients treated by the latter operation have no afferent loop, obstruction therein cannot be incriminated as an etiological factor.

Nevertheless, although uncommon, it would appear that afferent loop obstruction is not diagnosed as often as might be anticipated. It is interesting that more thought has not been given to the possibility of afferent loop obstruction since postoperative adhesions account for a large percentage of small bowel obstructions. In a recent follow-up study of 203 patients treated by gastrectomy for benign and malignant lesions of the stomach who were followed for one to five years, we encountered 5 cases of adhesive small bowel obstruction, 2 cases of acute afferent loop obstruction, one of whom subsequently also developed adhesive small bowel obstruction, and the 2 cases of chronic afferent loop obstruction reported here. Consequently, in our experience obstruction of the afferent loop has occurred almost as often as the more easily recognized obstruction of the distal small bowel. In all probability there are many patients who today are being treated by diet and antispasmodics, or simply being labeled neurotics because they represent no objective findings to explain their symptoms, who could be relieved by an operative procedure.

SUMMARY

Partial or recurrent obstruction of the afferent loop may occur following a Billroth II gastrectomy. This gives rise to postprandial nausea and vomiting of bile-stained fluid, unmixed with food. A simple test has been devised to aid in diagnosis predicated upon the fact that bile is normally present in jejunal contents. In patients with this disorder bile is absent from the jejunum during periods of symptoms, and the appearance of bile may be correlated with the relief of symptoms. The treatment consists of surgical relief of the obstruction. Case histories of 2 successfully treated patients are presented.

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THE INFLUENCE OF HYPOTHERMIA ON EXPERIMENTAL PERITONITIS

HENRY H. BALCH, MAJOR, MC USAR,* HOWARD E. NOYES, PH.D., AND CARL W. HUGHES, LIEUTENANT COLONEL, MC, USA, WASHINGTON, D. C.

WITH THE TECHNICAL ASSISTANCE OF W. BLALOCK, B.A., AND E. THIBODEAU, B.S.

(From the Division of Surgery, Army Medical Service Graduate School, Walter Reed Army Medical Center)

DEATH from peritonitis following penetrating or perforating abdominal injury has been reported within the last decade to range from 8.7 to 12 per cent.[2, 7] Likewise, a fatality rate of 4.9 to 9.4 per cent has been attributed to peritonitis resulting from perforated peptic ulcer or appendicitis.[2] This relatively favorable position has been attributed to many factors, including more precise indications for surgical intervention, the use of blood or other colloid supportive therapy, the control of intestinal distention, the correction of abnormalities in water and electrolyte exchange, and the use of antibacterial agents.

Under combat conditions, therapy may be less than optimal so that fulminating peritonitis is more prone to occur. The fatality rate in such patients is likely to be greater than that reported previously. It is important, therefore, to investigate alternative or additional methods in the therapy of peritonitis. The following report is concerned with the effect of hypothermia on experimental peritonitis in rats and the influence of dihydrostreptomycin on peritonitis in the hypothermic rat.

METHOD

White rats varying in weight from 120 to 180 grams were used. Peritonitis was produced by the standardized technique described by Pulaski and his associates.[6] Briefly, this consisted in incising the cecum at laparotomy and expressing feces into the peritoneal cavity. The cecal wound was not closed. Following suture of the abdominal wall, mucin was injected intraperitoneally. Mucin probably interferes with phagocytosis, thus permitting cecal bacteria to survive and multiply in the peritoneal cavity.

Anesthesia was induced with pentobarbital sodium, 40 mg. per kilogram of body weight. Additional anesthesia was given when needed by the intermittent administration of 16 mg. per kilogram of body weight of the drug. The intraperitoneal route was used originally but in later experiments anesthesia was induced by intramuscular administration and maintained by supplemental subcutaneous injections of pentobarbital because of more satisfactory anesthesia control.

Hypothermia was induced by placing anesthetized rats on dry towels in a metal pan which was partially immersed in ethylene glycol at 2° C. Under these conditions the animals' temperatures decreased from approximately 35° C. to 26° C. in about ninety minutes. When rectal temperatures fell below 25° C., the rats were removed from the "cold pan" and placed at room temperature until their temperatures reached 25° C. By alternately placing the

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*Present address: Washington Clinic, Wisconsin and Western Avenues, Washington, D. C.

animals in the "cold pan" and at room temperature it was possible to maintain animals within 2° C. of 25° C. Routine procedures were used in hematological studies of the peripheral blood and in aerobic and anaerobic bacteriologic studies of the peritoneal exudate.

RESULTS

Survival of Unoperated Rats Under Hypothermic Conditions.—The effect of hypothermia alone on a group of 24 anesthetized normal rats is recorded in

TABLE I. FATALITY RATE OF UNOPERATED RATS UNDER HYPOTHERMIC CONDITIONS (24 RATS)

TIME FROM ONSET OF HYPOTHERMIA (HOURS)	CUMULATIVE DEATHS	FATALITY RATE (PER CENT)
8	0	0.0
9	2	8.3
16	3	12.5
18	4	16.7
20	5	20.9
22	6	25.0
24	7	29.2

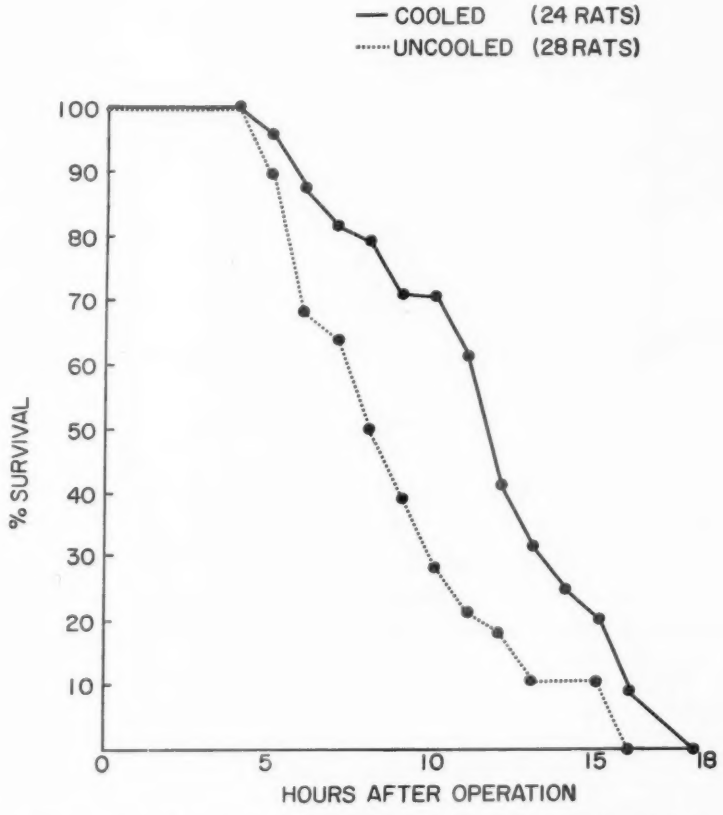

Fig. 1.—Survival of cooled and uncooled rats with experimental peritonitis.

Table I. All the rats survived eight hours, but subsequently there was a progressive increase in the fatality rate until, at the termination of the experiment (twenty-four hours), seven of the group had died (29.2 per cent). The data did not show whether death was the result of cold or due to the cumulative effect of pentobarbital sodium or to a combination of factors. It was apparent that

under the experimental conditions used, prolongation of the hypothermia state was associated with an appreciable fatality rate; however, at eighteen hours only 4 of 24 rats had died. None of the experiments involving infecton in this study lasted longer than eighteen hours, so it was concluded that the technique employed for cooling animals was satisfactory for this investigation.

Effect of Hypothermia on Survival With Peritonitis.—The survival of rats with experimental peritonitis was compared at room temperature and at 25° C. (Fig. 1). All of the uncooled rats were dead by the sixteenth postinfection hour and all of the hypothermic rats were dead by the eighteenth hour. Therefore, hypothermia alone did not prevent death from peritonitis. The mean survival time of the uncooled rats was 8.6 hours and of the hypothermic animals 11.4 hours. This difference, when analyzed by the method of Student, gave a value of 2.77 ($p<0.01$), which is significant. Fig. 2 records the mean temperatures of 20 rats from each group for approximately seven hours and demonstrates the definite difference in temperatures between cooled and uncooled animals. Preliminary observations had shown that pentobarbital sodium anesthesia alone will lower the mean temperature from 37° C. to about 35° C.

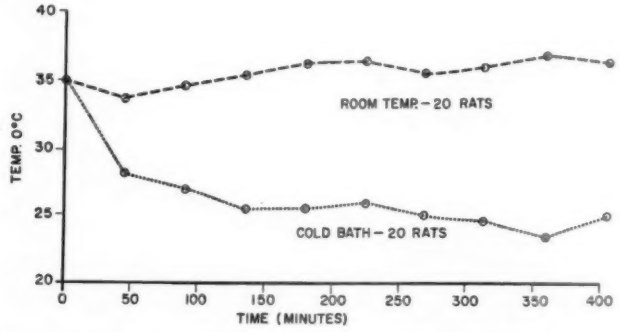

Fig. 2.—Rectal temperature of rats with experimental peritonitis (intramuscular plus subcutaneous anesthesia).

Bacteriologic Studies.—There was no significant difference in the bacterial species isolated from the peritoneal exudate of hypo- or normothermic rats. Total bacterial counts of the peritoneal exudate of infected rats were determined so as to show a possible effect of temperature on the rate of bacterial growth in vivo. The average numbers of bacteria recovered from the peritoneal cavities of cooled and uncooled animals sacrificed six hours after cecal perforation are

TABLE II. AVERAGE NUMBER OF BACTERIA RECOVERED FROM PERITONEAL CAVITIES OF RATS WITH EXPERIMENTAL PERITONITIS

GROUP	NUMBER IN GROUP	MEAN BACTERIAL COUNT $\times 10^6$	STANDARD ERROR MEAN
Cooled	42	3.45	0.95
Uncooled	42	6.04	1.27

compared in Table II. Although the mean appeared almost twice as great for uncooled as compared with cooled rats, this difference was not significant on statistical analysis. Comparison of total bacterial counts such as are recorded in Table II may be misleading, however, because of the difficulty encountered

TABLE III. AVERAGE BACTERIAL COUNTS PER MILLILITER PERITONEAL FLUID FROM RATS WITH EXPERIMENTAL PERITONITIS
(SIX HOURS POSTOPERATIVELY)

GROUP	NUMBER IN GROUP	MEAN BACTERIAL COUNT PER ML. PERITONEAL EXUDATE ($\times 10^6$)	STANDARD ERROR MEAN
Cooled	42	1.06	0.26
Uncooled	42	2.99	0.46

in removing all of the exudate from the peritoneal cavity of individual rats. If the bacterial counts from the same experiments are recorded as per milliliter of peritoneal exudate, then the differences are highly significant. These data are shown in Table III, where it may be seen that the bacterial count was almost three times greater in uncooled animals.

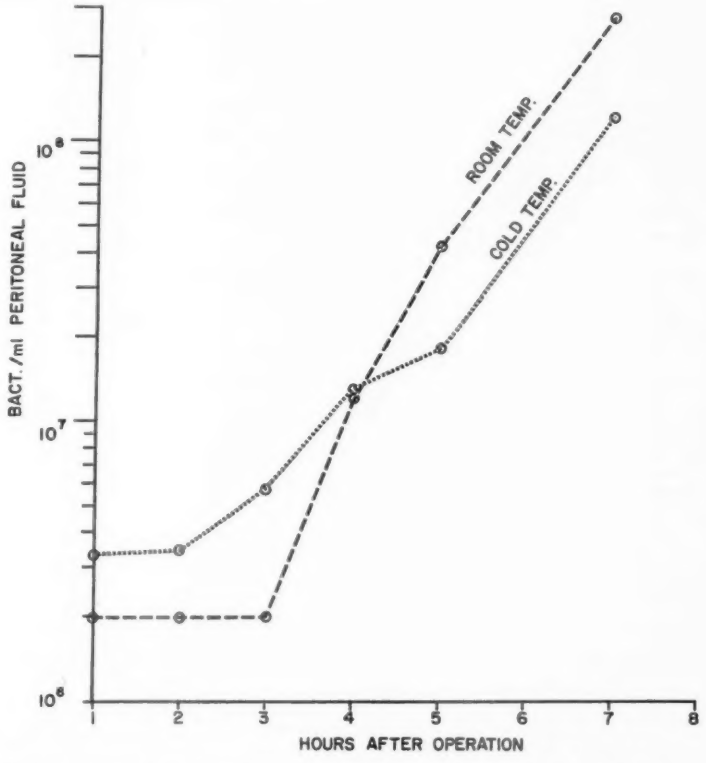

Fig. 3.—Bacterial counts of peritoneal fluid of untreated rats with experimental peritonitis.

Fig. 3 presents the bacterial counts per milliliter of peritoneal exudate from another experiment using cooled and uncooled rats. The counts were determined at hour intervals following cecal perforation (10 rats from each group were sacrificed every hour). The data did not appear to show that lowered temperature significantly reduced the rate of bacterial growth in vivo. These studies were not extended beyond seven hours because the increasing death rate precluded the obtaining of representative samples thereafter.

These bacteriologic studies did not, therefore, demonstrate conclusively that the rate of bacterial growth was delayed in the cooled animals. They did show

that bacterial growth occurred and was progressive at the hypothermic temperatures employed; eventually, bacteria reached what is probably a lethal concentration.

Leukocyte Counts.—No differences were found in the number of circulating leukocytes in the blood of normo- and hypothermic rats. Leukocyte counts of the peritoneal exudates were not satisfactory because the presence of mucin in the exudate rendered accurate identification and enumeration of leukocytes in unstained preparations difficult.

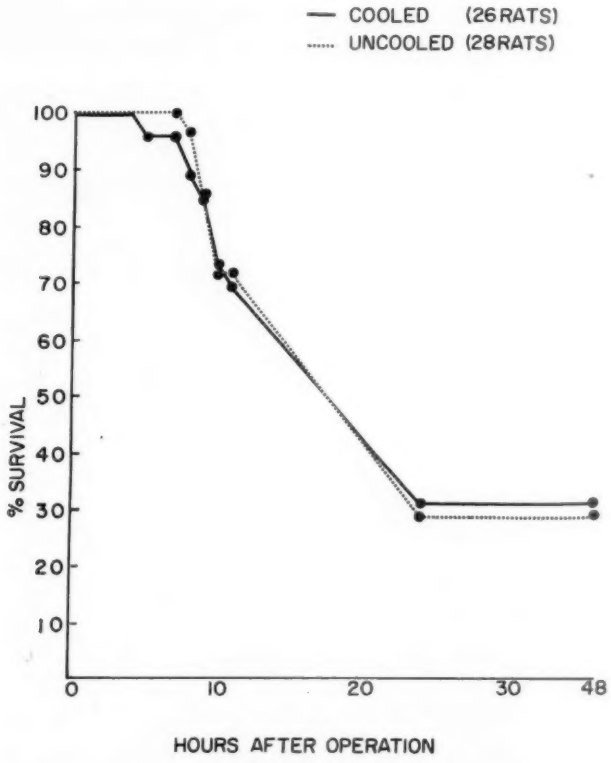

Fig. 4.—Survival of treated (intraperitoneal dihydrostreptomycin 40 mg. per kilogram of body weight) cooled and uncooled rats with experimental peritonitis.

The Effect of Dihydrostreptomycin on Survival From Experimental Peritonitis in Hypothermic Rats.—Previous studies by Pulaski and associates[6] showed that the administration of antibacterial agents in appropriate combination, dosage, and route led to a high survival rate from peritonitis in uncooled rats. When streptomycin was administered intraperitoneally, a moderate increase was noted in the survival of rats. Streptomycin is probably bactericidal to microorganisms growing at a slow rate,[4] as might occur under hypothermic conditions. Consequently, it was decided to use only dihydrostreptomycin in the present experiments in the hope of showing an additional effect due to hypothermia. Using the same techniques as before, all rats were treated with 40 mg. per kilogram of body weight of dihydrostreptomycin administered by the intraperitoneal route four hours following the induction of peritonitis. Eight of 28 uncooled animals

survived compared with 8 of 26 in the cooled group (Fig. 4). Mean survival time was increased to sixteen hours in both groups of animals. The combination of dihydrostreptomycin and hypothermia was not superior to the use of dihydrostreptomycin alone. Concurrent studies showed that if the handling of animals were eliminated, the administration of dihydrostreptomycin resulted in an increased survival rate over that obtained with animals that were manipulated. The hypothermic technique, however, requires handling for the recording and controlling of temperatures and the administration of pentobarbital.

DISCUSSION

The experiments reported in the present study showed that rats maintained at about 25° C. survived somewhat longer from experimental peritonitis than similar uncooled animals. This may be due in part to lower rate of bacterial multiplication at the lower temperature. However, at 25° C. bacteria multiply intraperitoneally and, eventually, all of the animals died.

We do not have data on the effect of cooling on the capacity of the rat to resist infection. It is possible that bacterial growth is delayed at lower temperatures in vivo, while defense against bacteria is also impaired. Harmon and his associates[5] found that polymorphonuclear leukocytes from rabbits and guinea pigs showed decreased phagocytic power for staphylococci as the environmental temperature was decreased from 42° C. to 22° C. The presence of mucin in the peritoneal exudate of our rats prevented studying the activity of the intraperitoneal leukocytes. However, it is likely that this was decreased as cooling increased.

It is of interest that Bruneau and Heinbecker[3] reported a delay in the destruction of hemolytic streptococci injected into dogs' forelegs which were subsequently cooled to 6° C. So long as cooling to 6° C. was maintained, the subcutaneous tissues of the dog failed to show the inflammatory response usually initiated by injection of the microorganism. The number of organisms present in the tissues remained approximately that injected. Animals whose forelegs were cooled up to forty-eight hours and then allowed to return to room temperatures showed a diminished number of viable bacteria forty-eight hours after the warming period was initiated. However, the destruction of bacteria did not approach that noted in dogs which had not been refrigerated at all.

It is likely that two interacting physiologic effects are present in hypothermic animals: a less rapid bacterial proliferation, but also a less efficient body defense. If such a system does operate in experimental infection under hypothermic conditions, then it seemed possible that the addition of a bacteriostatic or bactericidal chemotherapeutic agent to the system might be beneficial. Our data with the use of dihydrostreptomycin showed that the antibiotic prolonged life from peritonitis under hypothermic conditions, but it was equally effective in uncooled animals. In other words, when dihydrostreptomycin was used, there seemed to be no advantage in using hypothermia.

The use of cold to delay bacterial growth and tissue destruction has become a recognized method of treatment in diabetic and arteriosclerotic gangrene of

extremities. Such a procedure, when accompanied by the application of a tourniquet above the lesion, produces a physiologic amputation. Generalized symptoms of toxemia may abate for a time, permitting surgical amputation. Allen[1] has stated, however, that while an infected gangrenous limb may be frozen for days and the clinical condition improved, the limb must eventually be amputated. Cooling designed to reduce local metabolism in an infected part so that existing blood supply would suffice and healing follow has not been of value.

The data on experimental peritonitis from this paper suggest that cooling may be of value in slowing the development of infection but that, for recovery, additional therapeutic aids will be required. Under clinical conditions such an increase in the time factor might be of value where other immediate therapeutic aids were insufficient.

SUMMARY

1. Hypothermia was induced in rats by the administration of pentobarbital sodium and cooling to 25° C.

2. The mean survival time of hypothermic rats with acute peritonitis was increased to 11.4 hours as compared with 8.6 hours for uncooled rats.

3. The intraperitoneal administration of dihydrostreptomycin increased the mean survival time of cooled and uncooled rats with peritonitis to sixteen hours. Therefore, if the antibiotic was used, cooling did not appear to be of value.

4. Cooling to 25° C. did not interfere with the efficacy of dihydrostreptomycin.

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THE EXPERIMENTAL REPAIR OF URETHRAL DEFECTS

JOSE FIGUEROA-COLON, M.D., LEONARD BOGAEV, M.D., W. E. DEMUTH, M.D., A. C. BARET, M.D., KENNETH CARRINGTON, M.D., AND JOHN J. MURPHY, M.D., PHILADELPHIA, PA.

(From the Harrison Department of Surgical Research, Schools of Medicine, University of Pennsylvania, and the Department of Urology, Hospital of the University of Pennsylvania)

THE search for a suitable replacement for the urethra which has been destroyed by trauma or inflammation or is congenitally absent has continued since the first recorded efforts of Josserand in 1897.[1] Successful results have been reported using autogenous split-thickness skin grafts by Young and Benjamin,[2] with preputial skin by Pressman and Greenfield,[3] with autogenous bladder mucosa by Marshall and Spellman,[4] and with fresh cadaver urethrae by Bourque.[5] Isolated reports of success with tubes of foreign material such as silicone and polyethylene appear in the literature (DeNicola[6] and Foret[7]). The number of reported methods attest to the lack of uniform success in this problem. Current interest in grafts prepared by the "freeze-dried" techniques for arterial replacement and the recognition of the need for evaluation of some of the reported techniques stimulated the present study.

METHODS

Adult female mongrel dogs were used as experimental animals. In all except Group I, a urethral defect was created by excising a 2-cm. segment of the urethra immediately distal to the vesicourethral junction (Figs. 1 and 2). The following types of grafts were used to replace the excised segments: (1) Tubes of autogenous rectus fascia 2 to 3 cm. in length, fashioned by suturing an oblong piece of anterior rectus sheath loosely about a No. 12 or No. 14 Fr. catheter. (2) Segments of urethra taken from other animals and subjected to freeze-drying, according to the method of Lehr and Blakemore.[8] These were reconstituted by immersion in saline solution prior to use. (3) Segments of human and canine arteries treated by the freeze-drying technique and similarly reconstituted. (4) Homologous fresh urethral segments. (5) Tubes made of autogenous bladder mucosa, each 3 cm. in length and approximately 14 Fr. diameter.

A No. 12 or No. 14 Fr. catheter was placed through the graft to drain the bladder and splint the graft. The catheter was fastened by suture to the animal's vagina, leaving as little as possible protruding. Despite this precaution, the animals usually removed the catheters during the first week after operation. If this did not occur, the catheter remained in place from 7 to 21 days. The bladder and urethra were carefully examined at the time of death or sacrifice of the animal. Sections were made for histologic study. A normal animal was sacrificed to provide material for comparison of the histologic picture.

Six groups of animals were used in these experiments. In the first group, a preliminary exstrophy of the bladder was created by excision of the anterior two-thirds of the bladder and overlying abdominal wall. The bladder edge was sutured to the abdominal wall defect with 00 chromic catgut. A 2 cm. segment of the urethra was excised and subjected to the freeze-drying technique. At intervals ranging from 86 to 142 days postoperatively, the exstrophy was closed with a fascial graft from a rectus sheath of the same animal, as described by Baret and associates.[9] The segment of urethra previously prepared by freeze-drying was reconstituted in saline solution and sutured to the newly created vesical neck with fine arterial silk. The distal end of the segment was brought

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Fig. 1.

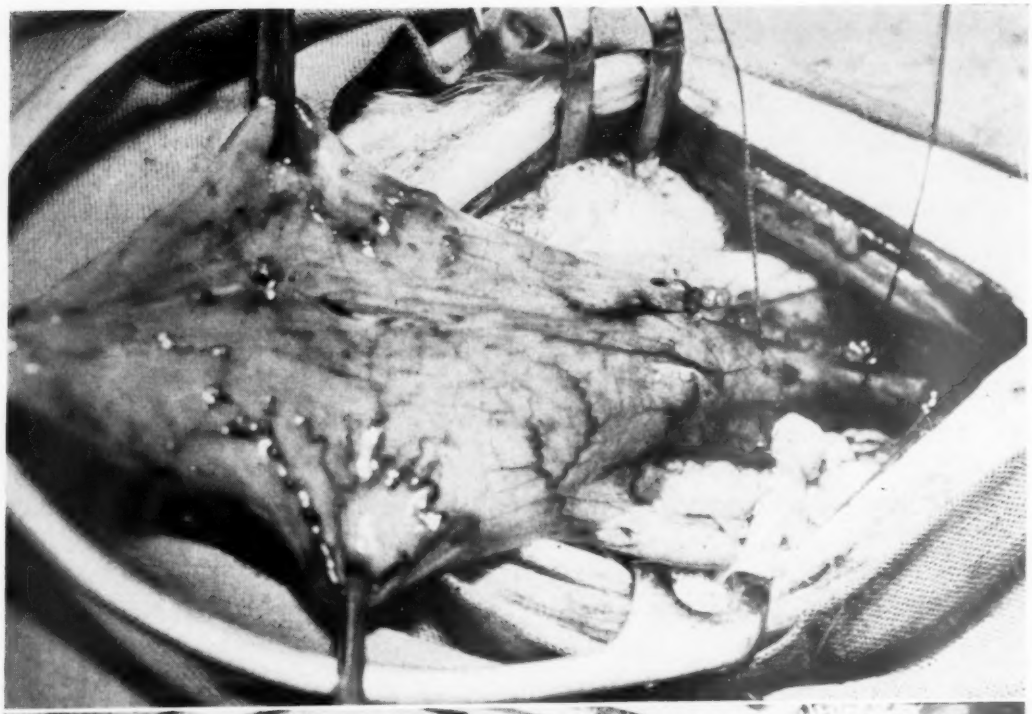

Fig. 2.

Fig. 1.—Exposure of bladder and urethra. Segment of urethra to be excised shown between sutures.

Fig. 2.—Urethral segment replaced with a fresh autogenous bladder mucosal graft.

out through a subcutaneous tunnel just above the symphysis pubis. The animals were sacrificed between 13 and 79 days after this operation (Table I). Examination of the graft site revealed that the entire graft had become necrotic by the thirteenth day, and complete obliteration by stricture had occurred at 22 and 79 days in the other animals. Similar changes to those found by Baret and co-workers were noted in the exstrophy repair. Moderate to severe hydronephrosis had developed in two animals and bladder calculi were found in one animal.

TABLE I. EXSTROPHY-FASCIAL GRAFT FREEZE-DRIED URETHRAL GRAFT

ANIMAL NO.	NECROPSY DAYS	RESULT
1,054	13	Necrosis of graft
723	22	Stricture entire graft
772	79	Stricture entire graft

Group II consisted of three animals in which the excised segment of urethra was replaced by an homologous urethral segment subjected to the freeze-drying technique. These dogs were sacrificed at 29, 30, and 44 days postoperatively (Table II). In the

TABLE II. FREEZE-DRIED HOMOLOGOUS URETHRAL GRAFT

ANIMAL NO.	NECROPSY DAYS	RESULT
1,171	29	Stricture entire graft
1,242	44	Stricture at junction of bladder-graft
894	30	Necrosis of graft

animal sacrificed at 29 days, there was evidence of stricture over the entire length of the graft. The lumen was small but not completely obliterated. The bladder, however, appeared dilated and very thin-walled. In the animal sacrificed at 30 days, the entire urethral graft appeared to have sloughed. At 44 days, a definite contracture was found at the vesicourethral junction, the lumen having narrowed to No. 8 Fr. The remainder of the graft appeared viable and was lined with mucosa. The bladder was very thick and trabeculated.

In Group III five animals received a graft made of human or canine freeze-dried arterial segments. These dogs were sacrificed or died at 4, 13, 48, 98, and 148 days postoperatively (Table III). In the animal which died at 4 days postoperatively, there

TABLE III. FREEZE-DRIED ARTERIAL GRAFT

ANIMAL NO.	NECROPSY DAYS	RESULT
66	4	Graft intact; lumen patent
1,241	13	Necrosis of graft
67	48	Stricture entire graft
523	98	Stricture at distal anastomosis
522	174	Stricture at junction of bladder-graft

was no apparent change in the caliber of the urethra or graft. At 13 days, the graft appeared to have sloughed and a vesicovaginal fistula was present. At 48 days, the animal showed a stricture of the entire length of the graft. At 98 days, there was a marked stricture at the distal anastomotic line, and in the one followed for 174 days, a vesicourethral stricture was present. In these latter two animals, the remainder of the graft appeared lined with mucosa, and the lumen, although somewhat narrowed, was not obliterated.

Group IV consisted of five animals with a graft made from the anterior rectus sheath (Table IV). Two animals died at 3 and 8 days, respectively, postoperatively. There was no change apparent in the caliber of the urethra in these animals. An animal sacrificed at 25 days showed no evidence of stricture and the anastomosis appeared intact and the

TABLE IV. AUTOGENOUS FASCIAL GRAFT

ANIMAL NO.	NECROPSY DAYS	RESULT
96	3	Graft intact; lumen patent
94	8	Graft intact; lumen patent
186	25	Graft intact; lumen patent; bladder distended
185	34	Stricture at junction of bladder-graft
184	43	Stricture, entire graft

lumen patent, but the bladder was dilated. At 34 days, the site of junction of the graft and bladder neck had greatly narrowed and, at 43 days, the entire length of the urethral graft had become narrowed and fibrotic.

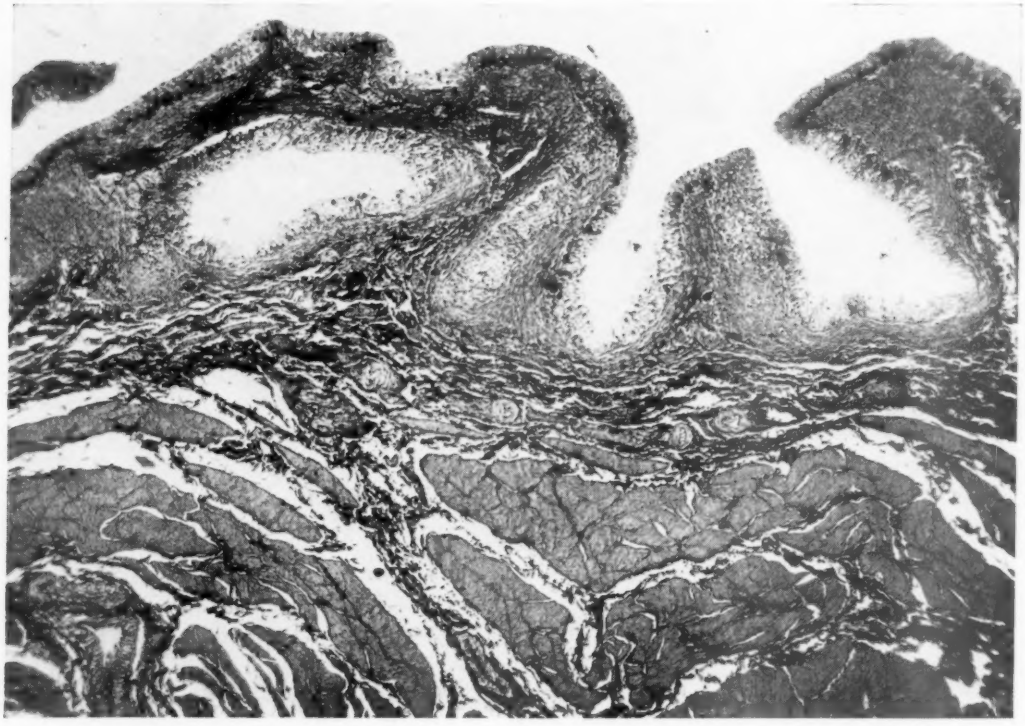

Fig. 3.—Normal histologic appearance of vesicourethral junction.

TABLE V. HOMOLOGOUS FRESH URETHRAL GRAFT

ANIMAL NO.	NECROPSY DAYS	RESULT
279	23	Stricture entire graft
521	24	Graft intact; lumen patent
307	73	Stricture entire graft
281	127	Stricture at junction of bladder-graft

Homologous urethral segments were used as grafts in Group V (Table V). Four animals made up this group. The first animal was sacrificed 23 days postoperatively. A filiform stricture involving the entire graft was found with a greatly dilated bladder above it. At 24 days, the catheter was still in place in the graft, and the lumen of the urethra appeared normal. At 73 days, a definite stricture of the entire graft had occurred. At 127 days, a bladder-neck contracture was present and there was secondary calcification at the proximal anastomotic line.

Autogenous bladder mucosal grafts were used in the three animals comprising Group VI (Table VI). These animals were sacrificed at 20, 39, and 140 days postoperatively.

Fig. 4.

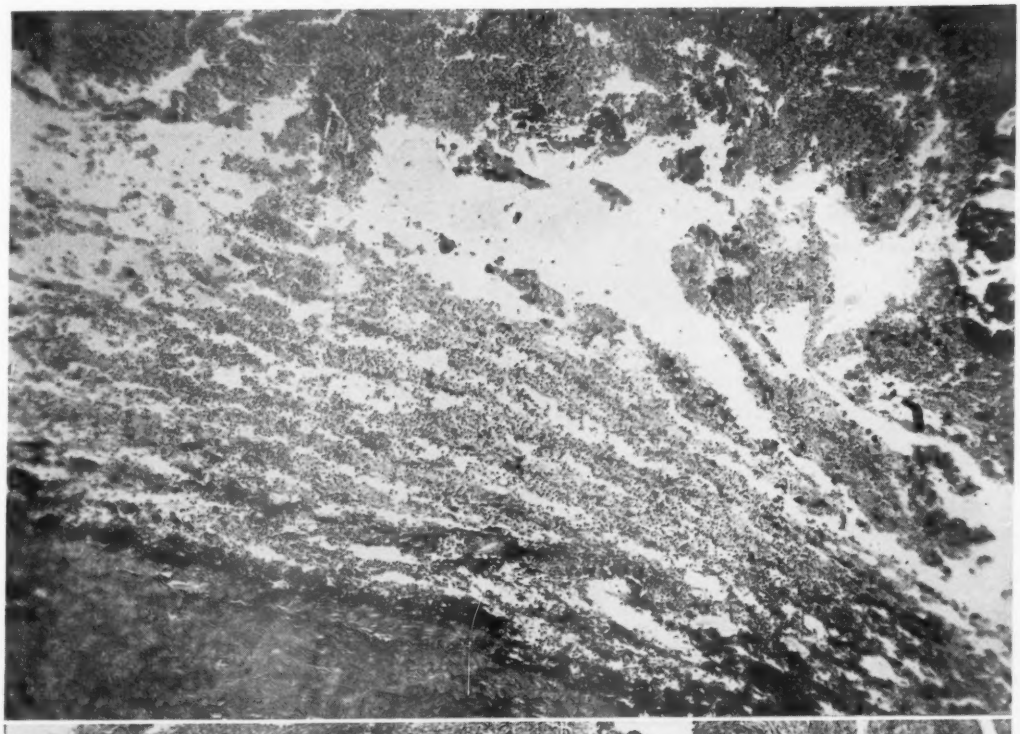

Fig. 5.

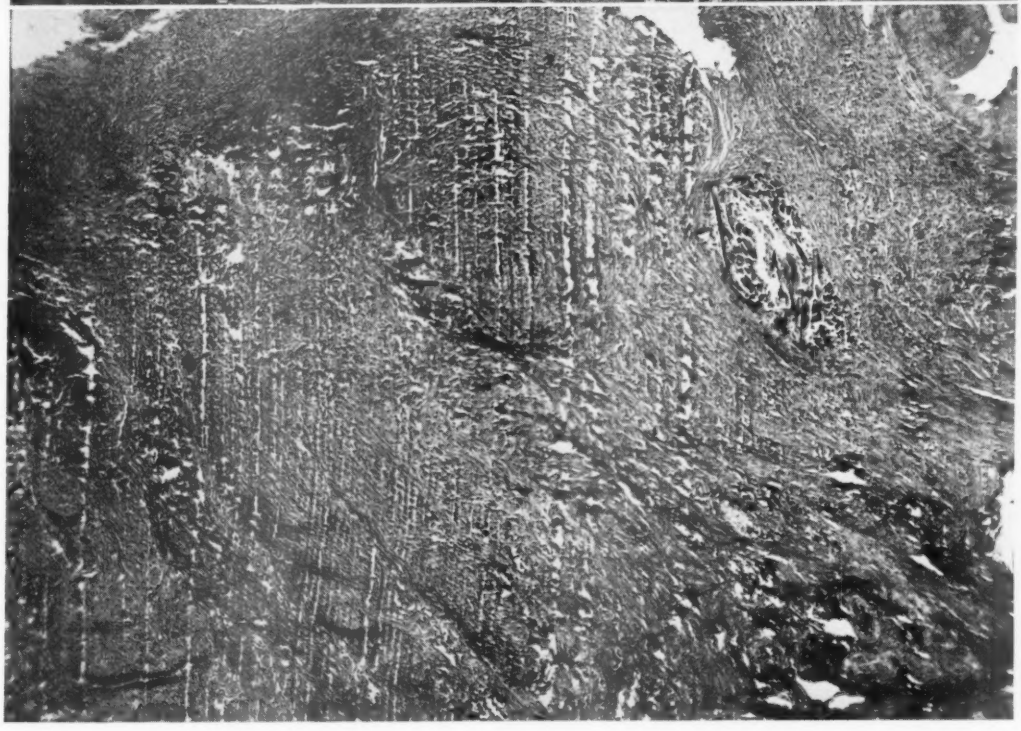

Fig. 4.—Marked inflammatory response to freeze-dried arterial graft (4 days).
Fig. 5.—Extensive fibrosis about freeze-dried urethral graft (44 days).

TABLE VI. AUTOGENOUS FRESH BLADDER-MUCOSAL GRAFT

ANIMAL NO.	NECROPSY DAYS	RESULT
893	20	Early stricture, entire graft
225	39	Necrosis of graft
618	140	Stricture at junction of bladder-graft

The first animal, sacrificed at 20 days, revealed a viable-appearing graft which was somewhat narrowed in its entire extent. In the animal sacrificed at 39 days, part of the graft had sloughed and there was infection and scarring of this area. In the animal sacrificed at 140 days, there was marked contracture at the vesicourethral junction with a huge dilated bladder proximal to this.

Fig. 6.—Moderate inflammatory response and fibrosis at vesicourethral suture line. Fresh bladder mucosal graft (20 days).

DISCUSSION

In the experimental animal, the repair of urethral defects with tissues from other animals of the same species results in a marked inflammatory reaction to the graft with subsequent fibrosis and contraction. Tissues preserved by the freeze-drying technique, both from the same species and other species, produce a similar response. Figs. 3, 4, and 5 demonstrate the histologic appearance of such grafts with marked inflammatory response and subsequent fibrosis as compared with the normal urethral picture. Fascia and bladder mucosa from the same animal appear to produce less inflammatory response and subsequent fibrosis, as shown in Fig. 6. Fibrosis and stricture formation at the sites of anastomosis of the grafts to normal tissues is a com-

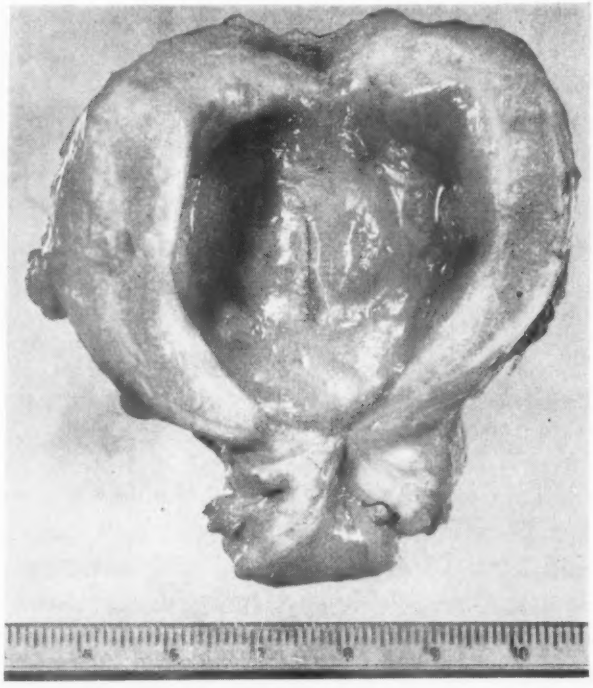

Fig. 7.—Obstruction due to severe contracture at bladder neck. Freeze-dried arterial graft (98 days).

mon occurrence (Fig. 7). Even though the graft may be viable, sufficient inflammation is produced to result in a functional obstruction in most cases.

CONCLUSIONS

1. The use of tissues, including urethra itself, whether preserved or fresh, as urethral substitutes is hazardous.

2. Failure of the graft may be due to immediate necrosis, delayed fibrosis and contraction due to marked inflammatory response, or stricture formation at the points of anastomosis to normal tissues.

3. Autogenous grafts of fascia or bladder mucosa appear to have some promise as evidenced by the production of less inflammatory response.

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VENTRICULAR FIBRILLATION IN DOGS AFTER SUDDEN RETURN OF FLOW TO THE CORONARY ARTERY*

WILLIAM H. SEWELL, LIEUTENANT, MC, USNR,** DOUGLAS R. KOTH, LIEUTENANT, JUNIOR GRADE, MC, USNR, AND CHARLES E. HUGGINS, LIEUTENANT, JUNIOR GRADE, MC, USNR, BETHESDA, MD.

(Naval Medical Research Institute, National Naval Medical Center)

INTRODUCTION

DURING experiments on temporary coronary arterial occlusion, ventricular fibrillation has been observed to occur within several seconds after the restoration of blood flow. This complication was noted in some of the dogs used for physiologic and pathologic studies by Tennant and Wiggers[1] and by Blumgart and co-workers.[2] The latter reported that 4 out of 34 dogs developed ventricular fibrillation within a few seconds after sudden release of a coronary artery. Swan and his group[3] and Bigelow and co-workers[4] reported fibrillation shortly after circulation was restored in dogs following its interruption under hypothermia.

We noted a similar phenomenon in a series of animals in which the anterior descending coronary artery was occluded temporarily. During operations for insertion of a polyethylene tube into this vessel, some of the animals developed ventricular fibrillation within twenty seconds after sudden restoration of the arterial flow.

Because of the increasing number of experimental and clinical procedures which require temporary interruption of the coronary flow, this hazard appears worthy of emphasis.

METHODS

In a preliminary report, a technique was described for the production of a gradual coronary occlusion by thrombus formation in a section of polyethylene tubing inserted into the anterior descending artery of the dog.[5] All of the 42 dogs in which this tube has been successfully inserted to date are included in the present analysis.

The first 6 dogs of the series were anesthetized with Pentothal, and the latter 36 with Nembutal. One hundred per cent oxygen was supplied under intermittent positive pressure by a respirator of our own design. The chest was entered through the bed of the left fifth rib, using aseptic technique.

A 2 to 3 cm. segment of the anterior descending coronary artery was dissected free and all branches ligated. In all of the dogs, the area of dissection extended to within 1 cm.

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*The opinions or assertions contained herein are the private ones of the writers and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

**Present address: Albany Hospital, Albany, N. Y.

of the bifurcation of the left coronary artery. The main septal branch was not ligated, even though encountered in some of the cases.

Potts patent ductus clamps were placed on the artery at the extremes of the dissected segment. A transverse incision was made in the center of it, and a section of polyethylene tubing approximately 7 mm. long was inserted into the lumen and tied in place. The period of occlusion was from three to thirty-eight minutes.

In the last 25 dogs of the series, a hole was made in the occluded segment with a small needle near the distal clamp. The proximal clamp was removed first and any air remaining in the vessel was forced out through this hole by the arterial pressure.

The distal clamp was removed suddenly in the first 31 operations. In the latter 11, a loop of 3-0 silk was placed around the artery proximal to the polyethylene tube. The heart was observed carefully after removal of the clamps, and the vessel was immediately occluded by tension on this loop if an extrasystole occurred. It was released after about twenty seconds but occluded again if necessary. In some cases, this cycle was repeated five or six times over a period of approximately five minutes before normal rhythm was maintained.

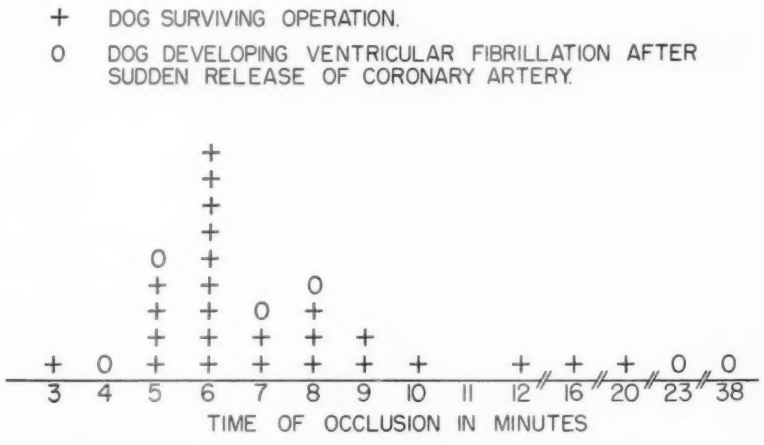
+ DOG SURVIVING OPERATION.
O DOG DEVELOPING VENTRICULAR FIBRILLATION AFTER SUDDEN RELEASE OF CORONARY ARTERY.
3 4 5 6 7 8 9 10 11 12 16 20 23 38
TIME OF OCCLUSION IN MINUTES

Fig. 1.—The relationship between the time of occlusion of the coronary artery and the development of ventricular fibrillation after sudden return of the blood flow.

RESULTS

Ventricular fibrillation developed within twenty seconds after release of the artery in 6 of the 31 dogs in which the flow was restored suddenly. In the 6 fatal cases, there was a normal rhythm for approximately the first five seconds, followed by extrasystoles. They were single at first and then occurred in short runs. Ventricular tachycardia ensued and progressed rapidly to ventricular fibrillation.

There appears to be no correlation between the length of time the artery was occluded and the occurrence of fibrillation after release (Fig. 1). Neither was there any significant relationship to the presence or absence of intact branches proximal to the occlusion.

When the blood flow was restored intermittently, as was done in 11 dogs, there was no fibrillation.

DISCUSSION

Large quantities of air in a coronary artery are known to produce ventricular fibrillation. However, small but visible quantities were forced distally into several of the surviving dogs without producing arrhythmia. Air was completely removed from the polyethylene tube in 4 of the fatal cases in the present series. This maneuver was not used in the other 2. It is possible that air embolism could have caused the death in the latter, although the air release hole was not made in 15 other dogs, all of which survived. In Blumgart's[2] study of temporary coronary occlusion, no opening was made in the vessel, so there was no possibility of the entrance of air.

In work not included in this report, defibrillation has been successful in animals anesthetized with Pentothal but nearly always failed in those under Nembutal. Vigorous electrical defibrillation and resuscitative measures were attempted on all of the hearts which fibrillated. Success was not achieved in any of the cases in the presently reported series, despite the fact that 2 of the dogs were under Pentothal.

The processes leading to fibrillation after the return of coronary flow are not understood. However, there are several pertinent observations in the literature.

A fall in pH and a rise in lactic acid level in the myocardium of dogs with coronary occlusion were demonstrated by Moore and Greenberg.[6] Evidence of carbon dioxide accumulation in the heart, along with a fall in the pH, during interruption of the circulation under hypothermia was reported by Swan.[3] He believed that the rapid return of the abnormally high carbon dioxide tension and the low pH toward normal may well have been a cause of fibrillation in his animals. Further evidence that these changes in blood chemistry can cause ventricular fibrillation was presented by Brown and Miller.[7] They observed arryhthmias in all, and ventricular fibrillation in 11 out of 15 dogs, immediately after a rapid reduction of an abnormally high carbon dioxide tension in the alveoli. When the carbon dioxide concentration was reduced slowly in two dogs, no arrhythmias occurred. It seems possible that a similar process was operating in the presently reported series.

SUMMARY

The anterior descending coronary artery of dogs was temporarily occluded for the insertion of a polyethylene tube. Sudden return of the arterial flow after completion of this intubation was followed by ventricular fibrillation within twenty seconds in 6 of 31 animals. Intermittent release over a period of several minutes was associated with no fatalities in 11 dogs.

These observations would seem to justify exercise of caution during the restoration of temporarily interrupted coronary flow in various types of procedures.

We wish to acknowledge the skilled operating room management and technical assistance of the following men during the experiments included in this review: R. M. Barber, HM3; K. G. Gibbons, HM3; S. T. Scott, HM2; C. J. Waido, HM3, and H. E. Warford, HM3. Students from the Tissue Bank School of the Naval Medical School assisted during the operations.

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A SIMPLIFIED TECHNIQUE FOR DIVISION AND SUTURE OF THE PATENT DUCTUS ARTERIOSUS

LEWIS H. BOSHER, JR., M.D., RICHMOND, VA.

(From the Division of Thoracic and Cardiovascular Surgery, Department of Surgery Medical College of Virginia)

SINCE the first successful surgical obliteration of the patent ductus arteriosus by Gross[7] in 1938, surgeons have frequently argued the advisability of attempting complete division of this channel.

The chief proponents for division and suture have been Gross,[8] Potts,[12] Jones,[11] Wangensteen,[14] Conklin,[3] Glenn,[6] and Freeman,[5] whereas Blalock,[2] Scott,[13] Holman,[10] and Bigger[1] have presented rather convincing statistics which show that ligation or suture ligation, when properly performed, only infrequently fails. Failure may result from incomplete closure of the ductus, recanalization when sutures cut through, or re-establishment of fistula due to infection and erosion around sutures.

On theoretical grounds alone it is generally agreed that division of the ductus is desirable. It is for this reason that we present a technique which we now employ in most cases and which seems to offer a high degree of safety.

Division and suture of a short, wide ductus presents a real technical challenge. This is especially so in the adult where the aorta and pulmonary artery are large and the exposure between these vessels is limited. If, in addition, a sclerotic, friable ductus is encountered by the surgeon, wide experience and great skill will be required to guide him through the procedure without disaster. It is in just such difficult situations as these that ligation or suture ligation of the ductus may also fail.

In presenting the following technique for division and suture of the patent ductus we make no claim for originality. Almost all of the steps have been previously described by individual surgeons. However, we have attempted to synthesize the most useful technical maneuvers and have added a few modifications. Our technique is applicable to most cases of patent ductus; the necessary variations for dealing with the remaining cases will be mentioned in the discussion.

TECHNIQUE

Through a standard lateral approach, the pleural space is entered through the left fourth intercostal space in the infant and child and through the bed of the fifth rib in the adult. The second assistant retracts the upper lobe

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downward. Later, to provide the surgeon with maximum exposure beneath the ductus, the first assistant sweeps the pulmonary artery forward by retracting the lung anteriorly.

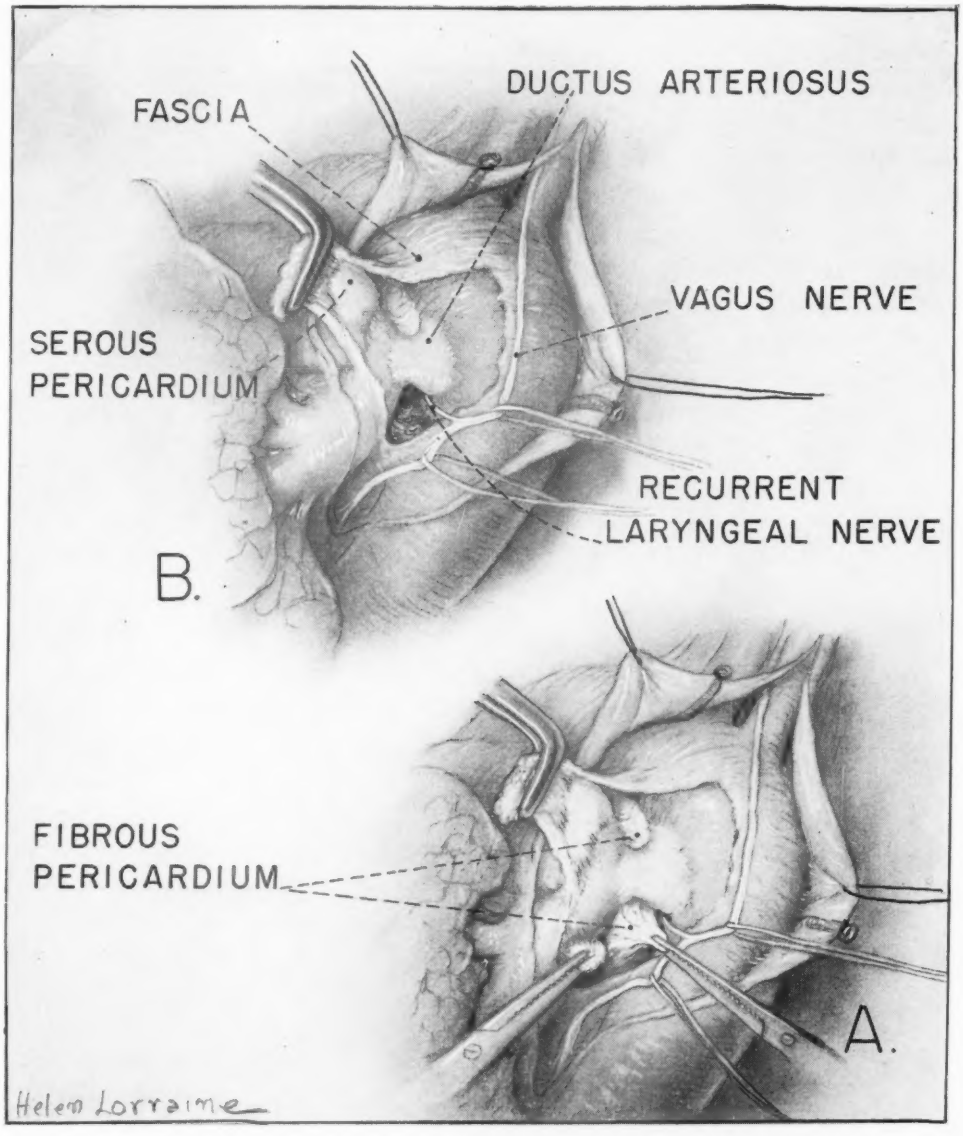

Fig. 1.—*A*, Exposure of the region of the ductus is shown after wide incision of the pleura. Vagal branches to the pulmonary artery have been divided. The perivascular sheath over the aorta has been incised and the fibrous tissue and lappet of serous pericardium dissected off the ductus onto the pulmonary artery. *B*, The recurrent nerve has been carefully exposed and removal of fibrous pericardium from beneath the ductus started.

After the chest is entered the highest intercostal vein is divided and the mediastinal pleura incised. The pleural incision is extended quite far inferiorly behind the hilum while avoiding injury to the nearby vagus and phrenic nerves. By retracting the lung forward, one brings into sharp relief the vagal branches to the left pulmonary artery and hilum. These vagal

branches are divided so as to expose fully the region inferior to the ductus. This likewise permits easy identification of the recurrent nerve in its constant position encircling the ductus. Excessive traction on the vagus nerve above the recurrent branch may cause injury to the delicate recurrent nerve fibers. Excision of several lymph glands is often required during the dissection.

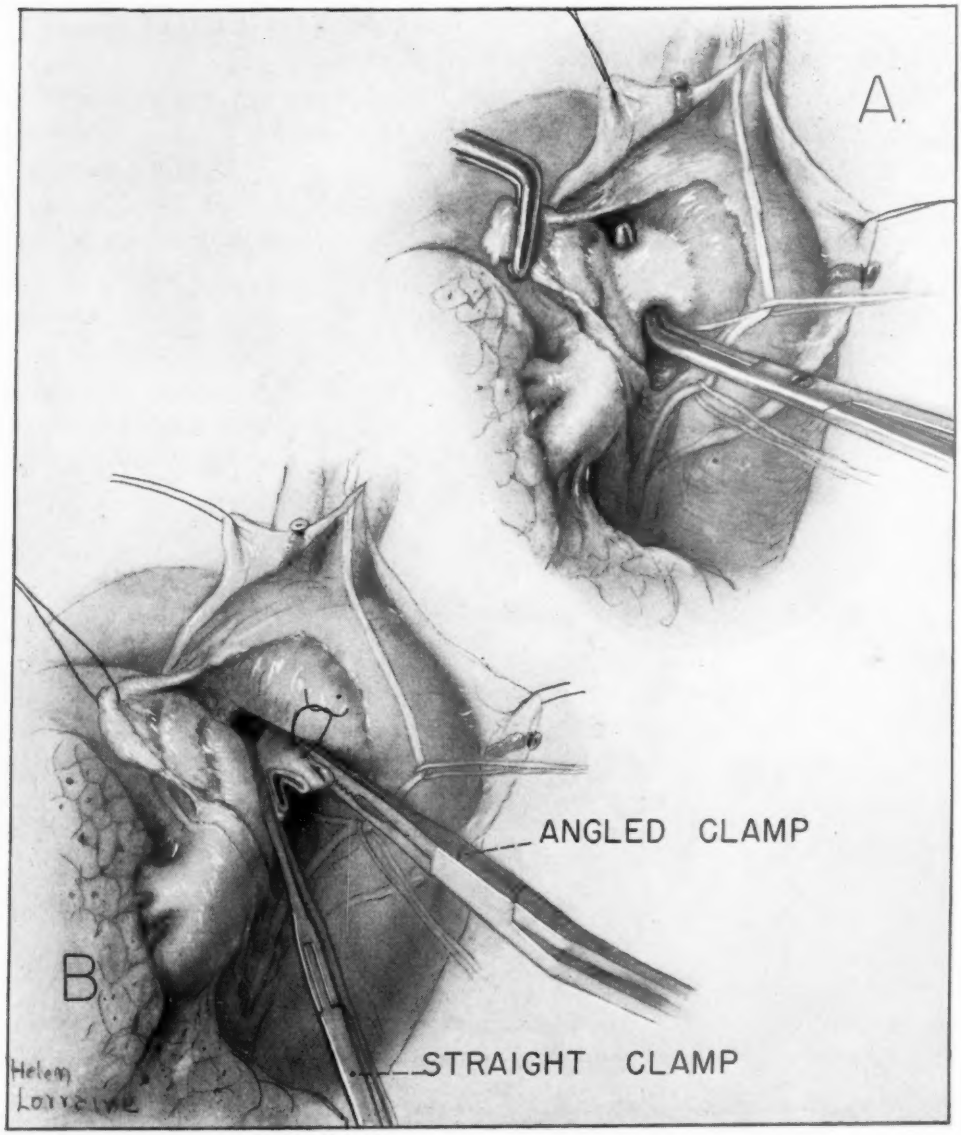

Fig. 2.—*A*, Further meticulous removal of fibrous pericardium permits passage of a clamp beneath the ductus. *B*, The angled and straight Potts ductus clamps are applied at the extreme ends of the ductus. (The Potts bronchus clamp is now used instead of the angled ductus clamp.) Transection is started, leaving a wide aortic cuff. Interrupted mattress sutures are inserted in stepwise fashion as the ductus is progressively divided.

Isolation of the ductus proceeds easily if the surgeon then incises completely through the perivascular sheath overlying the aorta to enter the proper plane. With sharp scissors dissection, the fibrous tissue and soon the

lappet of serous pericardium which extends over the ductus for a variable distance are stripped back well onto the pulmonary artery (Fig. 1, *A*). It is very important, however, to avoid removing adventitia and thus weakening the ductus wall. While these maneuvers are carried out the recurrent nerve is isolated as it swings beneath the ductus.

Final preparation of the ductus for division entails meticulous excision or sectioning of all fibrous pericardium from the region around and beneath the ductus, especially in the angle between the ductus and pulmonary artery (Fig. 1, *B*). This is the most tedious and yet the most important part of the dissection. Occasionally, while dissecting out the fibrous pericardium, the pericardial sac will be entered. Such a wide dissection is required to yield maximum length of ductus together with a portion of the pulmonary artery and to permit safe application of the Potts clamps. A right-angle clamp should not be passed beneath the ductus until the fibrous tissue has been removed in part, since forced traction may tear a fragile ductus (Fig. 2, *A*). Gentle elevation of the ductus by an umbilical tape facilitates dissection beneath the ductus. Exposure beneath the ductus is further improved if the first assistant gently retracts the pulmonary artery with a gauze pledget, thus allowing the surgeon always to dissect under direct vision. Before final clamping, the ductus is temporarily occluded for several minutes and the physiologic effects observed.

Optimum placement of the Potts clamps is accomplished in the following manner. The first assistant tents up the pulmonary entrance of the ductus using two gauze pledgets on the pulmonary artery, the second assistant rolls back the recurrent nerve on the aorta with a third pledget, and the surgeon, while elevating the mid-portion of the ductus with a right-angle clamp, places an angled Potts clamp across the ductus well back against the aorta. Formerly we used the angled Potts ductus clamp, but more recently have learned that much better exposure is provided for suturing if the Potts bronchus clamp with its more distal angulation is utilized on the aortic side of the ductus. Occasionally it may be helpful to apply the angled ductus clamp and bank the bronchus clamp behind this to gain the maximum length of ductus on the aortic side. The straight ductus clamp is applied across the opposite end of the ductus well onto the pulmonary artery. The Potts clamps must be occluded to the last rachet, and rachet locks are then clamped on (Fig. 2, *B*).

Division and suture of the two ends of the ductus is performed in a stepwise fashion. A short incision is initially made into the ductus with fine straight scissors at a point which leaves a wide cuff on the aortic side and usually, unless the ductus is long, a short cuff on the pulmonary artery side. This initial incision permits slight spreading of the clamps and facilitates placement of the first of three or four interrupted mattress sutures of 4-0 arterial silk close to the aortic cuff clamp. The ductus is progressively divided as the remaining interrupted sutures are inserted and tied (Fig. 3, *A*). This technique yields a wide aortic cuff, maintains the lips in eversion for the final

rows of sutures and completely protects the surgeon against possible slipping of the aortic cuff or retraction of the posterior wall of the ductus, which may be quite thin. Using 5-0 arterial silk, closure is completed with two rows of

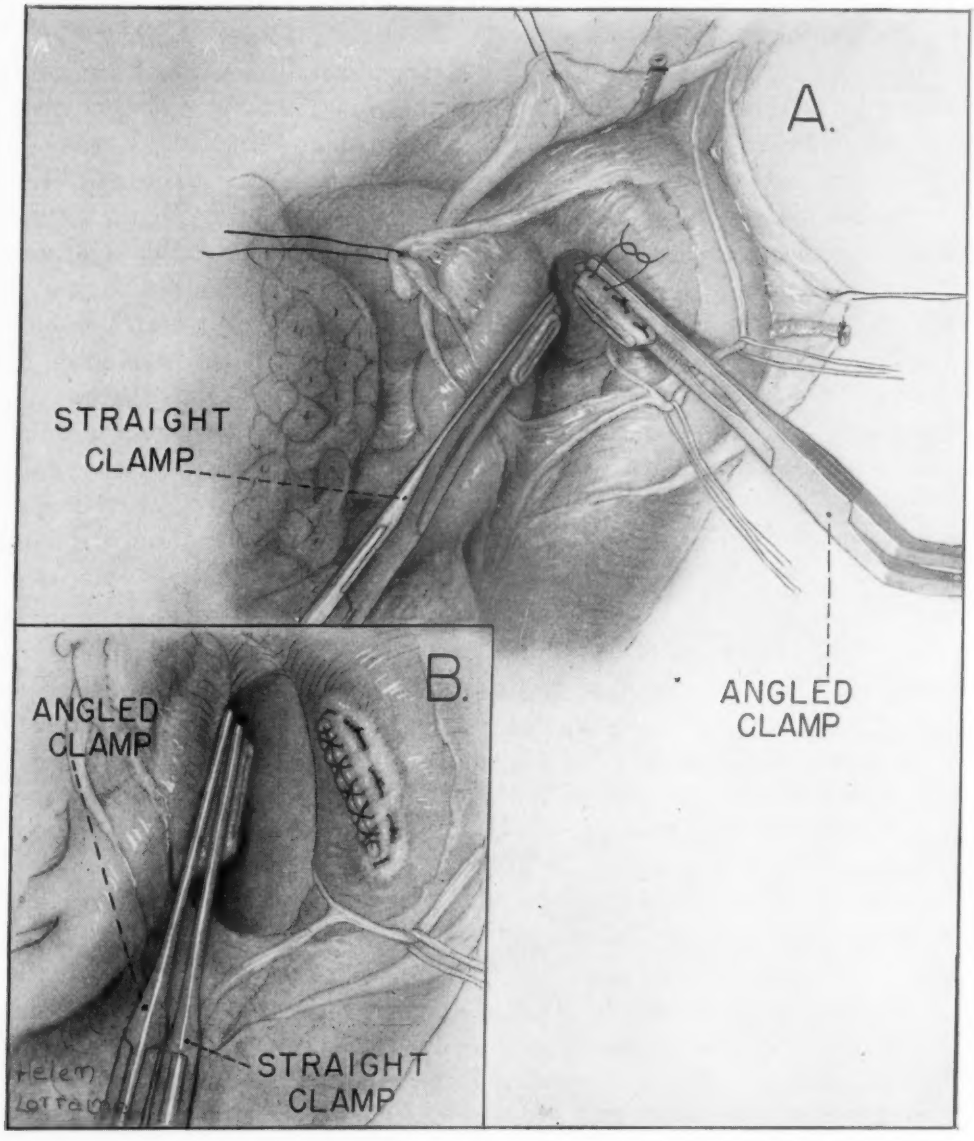

Fig. 3.—*A*, Complete transection of the ductus permits more adequate exposure, and closure of the aortic cuff is completed with two rows of continuous sutures or interrupted figure-of-eight sutures. *B*, Additional length can now be gained on the pulmonary cuff by placement of an angled clamp beyond the straight clamp.

continuous sutures or interrupted figure-of-eight sutures over the end of the aortic cuff. If these sutures are properly placed, no additional interrupted sutures will be necessary after removal of the clamp.

Once the ductus is completely transected, a more adequate pulmonary cuff may easily be gained by placement of an angled Potts ductus clamp behind

the straight clamp (Fig. 3, *B*). This may be done immediately, if two angled clamps are available, or later, after the aortic cuff is sutured and the angled clamp removed from it. If still more length is desired on the pulmonary artery cuff, the more sharply angled Potts bronchus clamp may be applied beyond the angled ductus clamp, but this additional step is rarely necessary.

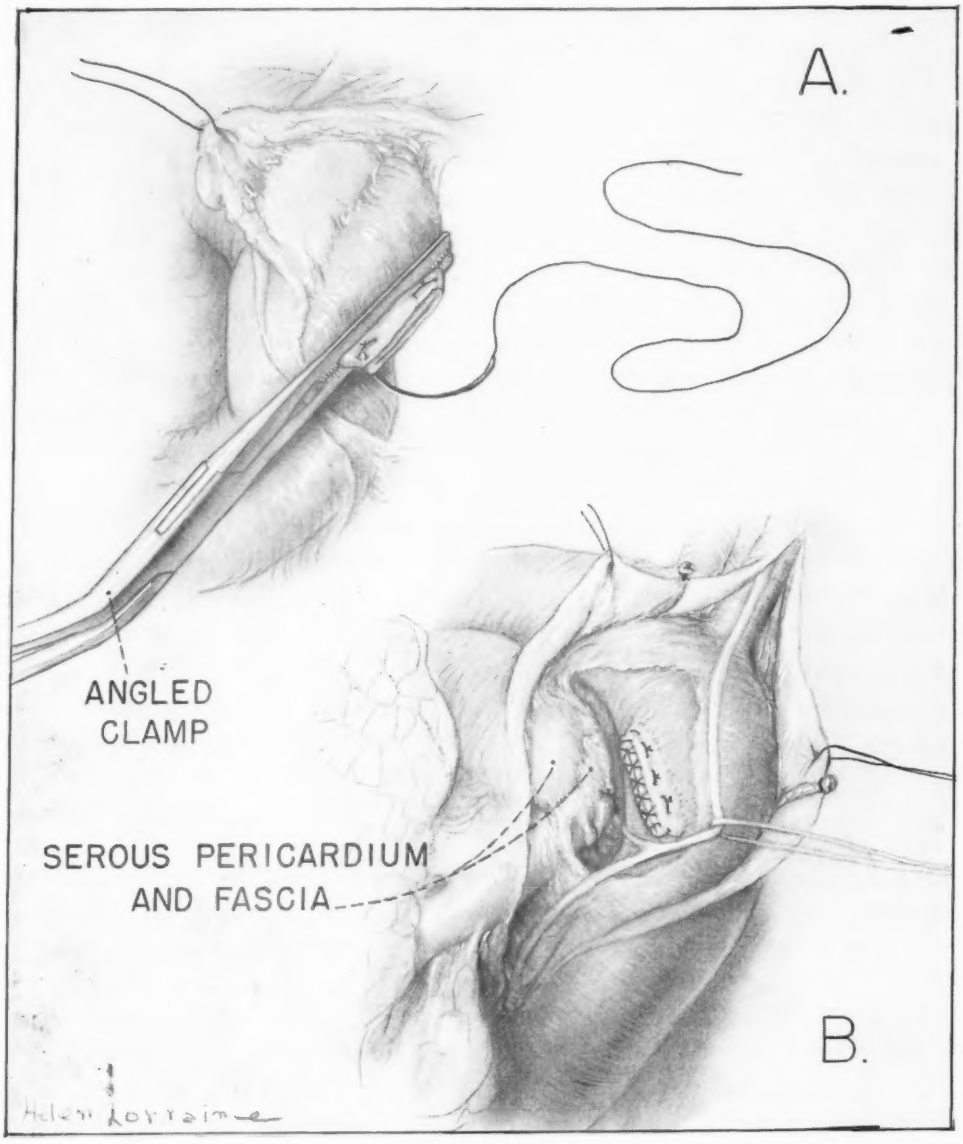

Fig. 4.—*A*, Closure of the pulmonary cuff is illustrated. *B*, The pulmonary cuff is covered by serous pericardium and fibrous tissue sutured down to the fibrous pericardium beneath the pulmonary artery.

For suturing the pulmonary cuff we have usually preferred an interrupted mattress suture at either angle to prevent inversion of the corners, followed by two rows of continuous sutures over the end of the cuff (Fig. 4, *A*). To protect against possible recanalization even after division of the

ductus, we have always covered the pulmonary artery cuff with the lappet of serous pericardium and fibrous tissue, suturing it to the fibrous pericardium beneath the pulmonary artery (Fig. 4, *B*).

The mediastinal pleura is sutured loosely. The pleural space is always drained by an intercostal tube, which is generally removed in twenty-four to forty-eight hours.

DISCUSSION

A number of disasters have been reported in the literature during attempted obliteration or division of the patent ductus. Hemorrhage has occurred during dissection of the undersurface of the ductus or has resulted from forceful spreading of the tissues beneath the ductus with a clamp. Injury to the pulmonary artery has occurred at a site of aneurysmal dilatation. Many of these disasters probably can be attributed to lack of exposure. Fatalities have occurred because clamps have slipped from the cuffs of the transected ductus. Such an event can result from inadequate removal of fibrous pericardium which prevents complete closure of the clamp. Clamps directly applied across a sclerotic, friable ductus such as may be encountered in an elderly patient or in the presence of endarteritis may cut through. Several instances are recorded in which infection developed around heavy sutures used for ligation, leading to the establishment of an endarteritis and a recurrence of the fistula.

In the technique described maximum exposure of the region around and beneath the ductus is provided by employing the lateral approach, incising the pleura quite far inferiorly behind the hilum of the lung, dividing the vagal branches to the pulmonary hilum and retracting the lung and pulmonary artery forward. The important steps in dissecting out the patent ductus have been lucidly described by Potts.[12] These include sharp (scissors) dissection; utilization of the proper plane of dissection, initially on the aortic side of the ductus; removal of all fibrous pericardial tissue about the ductus; and isolation of a considerable portion of the pulmonary artery as the serous pericardium is dissected back. We wish to stress the desirability of obtaining a wide aortic cuff at the expense of the pulmonary artery cuff, since additional length can always be gained on the pulmonary artery side after transection. Accidents relating to slipping of the cuff from the jaws of the clamp can be averted by the insertion of proximal mattress sutures during transection, a step described by Glenn,[6] who, however, prefers a continuous suture.

In the short, wide, but still elastic ductus encountered in the infant or child, no difficulty should be encountered in obtaining sufficient length to permit division and suture by the technique described previously. However, if the surgeon feels any anxiety in this regard, he should not hesitate to mobilize the aortic arch above and below the ductus as a safeguard in order to permit application of a partially occluding aortic clamp or double cross-clamping, as preferred by Crafoord.[4] Mobilization of the arch adds additional time to the operative procedure and increases slightly the risk of damaging the recurrent nerve. We have employed it rarely in the child but consider it a valuable ma-

neuver in the adult patient with a large aorta and pulmonary artery, and especially in those cases in which the ductus is sclerotic. This maneuver does in considerable measure facilitate dissection of the undersurface of the difficult ductus. After mobilization of the arch the surgeon may then elect to occlude the ductus with Potts clamps or to occlude the aortic end of the ductus with a partial occlusion clamp of the Potts-Smith type positioned about the aorta. In either instance the stepwise method of transecting the ductus will prove valuable. In the case with a sclerotic and brittle ductus, the application of a partial occlusion clamp probably offers the safest technique and may help to avoid the complication which caused our only fatality.

Out of forty-nine cases of patent ductus surgically treated by the author, thirty-six have been divided and sutured. Twenty-eight of the last thirty have been so managed. Partial aortic occlusion clamps have been utilized in seven cases and cross-clamping of the aorta in two additional instances in which technical difficulties arose.

There has been one operative death, the first case in the series: a 52-year-old woman with auricular fibrillation, cardiac failure, and tertiary syphilis. The aorta and pulmonary artery were tremendously enlarged and the ductus extremely brittle. Minimal manipulation in the region beneath the ductus produced hemorrhage. A partial occlusion clamp of adequate size was not available for use on the aorta. An initial attempt to ligate the ductus was abandoned because of hemorrhage and, upon clamping the ductus with Potts clamps, the ductus snapped away cleanly from the aorta. The aorta was cross-clamped, but attempts at suturing the orifice were futile, since all sutures promptly cut through under systemic pressure. This region was resected and the aorta anastomosed but, to accomplish this, a clamp had to be placed proximal to the left carotid artery. Cardiac arrest resulted upon final release of the aortic clamps.

No serious complications have occurred in the remaining forty-eight cases. Two instances of temporary left vocal cord paralysis have cleared completely and we believe these were related to excessive traction on the vagus nerve. There have been no recurrences in the entire group.

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Case Reports

FACTORS AFFECTING SPINAL CORD ISCHEMIA DURING AORTIC OCCLUSION

B. EISEMAN, M.D., AND W. B. SUMMERS, M.D., DENVER, COLO.

(From the Department of Surgery, University of Colorado School of Medicine, and the Denver Veterans Administration Hospital)

ALTHOUGH the operative excision and replacement of a diseased portion of the aorta is now a well-established technical procedure, ischemic damage to the spinal cord during the required period of aortic occlusion remains a constant threat. A number of techniques,[8, 27] including generalized hypothermia,[3, 13, 23-26] have been investigated in an attempt to minimize this problem. The purpose of this paper is to present a case of permanent spinal-cord damage following resection of an aneurysm of the descending thoracic aorta under hypothermia anesthesia, to emphasize the wide variation and tenuous nature of the vascular supply of the cord, and to discuss the various factors that affect the safe period of aortic occlusion.

CASE REPORT

P. C. S., a 32-year-old white man, suffered a gunshot wound of the left thorax in 1944, the bullet entering the sixth intercostal space anteriorly and lodging in the right lung field at the level of T 10 posteriorly. There was no mention of aortic involvement at the time of thoracotomy immediately following the injury. In August, 1954, following admission to the Denver Veterans Administration Hospital for investigation of an acute midline upper abdominal pain, he was found to have an aneurysm of the thoracic aorta extending from T 10 to T 11 (Fig. 1). On Aug. 3, 1954, the diseased aorta was resected and a lyophilized aortic homograft inserted, utilizing hypothermia (28° to 30° C.) anesthesia. Proximally, the aorta was clamped at the level of T 8 and the segmental arteries interrupted at T 9 in order to facilitate the anastomosis. The distal anastomosis was performed at T 12, the clamp being placed immediately proximal to the celiac axis between L 1 and L 2. Thus, the segmental arteries at T 9, T 10, T 11, T 12, and L 1 were severed. Due to the friable nature of the proximal aorta it was necessary to take down and revise the proximal anastomosis. This necessitated thoracic aortic occlusion for 63 minutes at the level of T 8.

Postoperatively the graft functioned well (Fig. 2) and vascular supply has continued to be excellent. There is, however, permanent severe motor deficit of both lower extremities which is related to ischemic damage to the anterior horn cells of the lumbosacral segments of the cord with maximal damage below the third lumbar level.

DISCUSSION

The limiting time of thoracic aortic occlusion is dictated by the resultant damage to the spinal cord, not irreversible changes in the kidney or abdominal viscera.[18] The wide and unpredictable individual variation in spinal-cord tolerance to aortic cross-clamping is disturbing and challenging to the vascular surgeon. Whereas one patient may tolerate over two hours of aortic occlusion

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Fig. 2.

Fig. 1.

Fig. 1.—Roentgenogram of abdomen showing preoperative aortic aneurysm at T 10.
Fig. 2.—Postoperative aortogram showing functioning homograft.

distal to the left subclavian artery,[21] another will suffer severe cord damage after less than forty minutes of cross-clamping.[10, 11, 15, 18] There are a number of factors affecting such spinal-cord damage and each must be considered individually in order to evaluate the problem properly, or to draw any significant conclusions from the mass of experimental and clinical data available in the literature concerning the safe period of aortic occlusion (Fig. 3, Tables I and II).

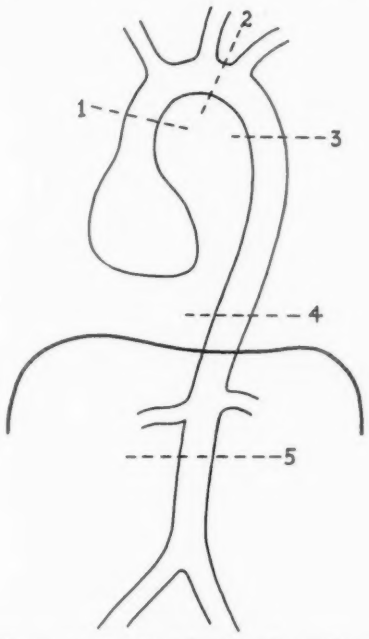

Fig. 3.—Diagram showing levels of aortic occlusion.

1. *The Arterial Blood Supply of the Spinal Cord and Its Variations.*—Since spinal-cord ischemia is the limiting factor in thoracic aortic occlusion it is of paramount importance to understand the anatomic distribution of the arterial supply of the normal spinal cord and to correlate the clinical occurrence of paraplegia following cross-clamping of the aorta with the known location of the major arterial channels.

TABLE I. AORTA OCCLUSION IN MAN

AUTHOR	LEVEL	TIME (MINUTES)	TEMP. (°C.)	PARALYSIS		DISEASE
Beattie[2]	2	41	37°	1/1	100%	Coarctation
DeBakey[13]	2	62	28°	0/1	100%	Aneurysm
Swan[25]	2	62	37°	1/1	100%	Coarctation
Bing[4]	2 & 3	Not stated	37°	1/23	4%	Coarctation
Cooley[10, 15, 18]	3	38-77	4 at 28° 8 at 37°	1/12	8% (transient)	Aneurysm
Lam[23]	3	39 (24 + 15)	37°	1/1	100%	Aneurysm (used shunt)
DeBakey[15]	4	45	37°	0/1	0%	Aneurysm
DeBakey[17]	5	90-120	37°	0/26	0%	Aneurysm and obliterative

The two most striking characteristics of the arterial supply of the cord[1, 6, 22, 28, 29] are its wide individual variation and the small number and size of the

TABLE II. AORTIC OCCLUSION IN DOGS

AUTHOR	LEVEL	TIME (MINUTES)	TEMP. (°C.)	PARALYSIS		DISEASE
Beattie[3]	2	60	37°	4/10	40%	
Beattie[3]	2	60	20-30°	?1/10	10%	
Blalock[5]	2	40-65	37°	21/43	50%	Artificially produced coaretation
Mahorner[24]	2	Not stated	37°	0/3	0%	
Carrel[7]	3 & 4	10-15	37°	Occasional (% not stated)		
Clatworthy[8]	3	30	37°	10/13	77%	
Gross[20]	3	4-15	37°	1/12	8%	
Gross[20]	3	23-60	37°	7/14	50%	
Owens[25]	3	20-30	37°	18/36	50%	
Owens[25]	3 & 4	90-180	23-26°	0/7	0%	
Owens[25]	4	90-180	23-26°	0/9	0%	
Pontius[13, 26]	3	60	37°	33/50	65%	
Pontius[26]	3	60	24-27°	0/47	0%	
Schafer[27]	3	30-50	37°	6/7	86%	
Lam[23]	Ligate all intercostals 3 & 4	0	37°	2/10	20%	

arterial channels supplying this vital structure. The most important artery to the cord is the anterior spinal artery, a vessel of varying size that runs up the midline of the cord anteriorly and sends branches into the substance of the cord. A smaller and less important pair of posterior spinal arteries lie on each side of the cord posteriorly and supply the posterior portion of the posterior columns and a part of the posterior horn (Fig. 4). The remaining portion of the cord is vascularized by the anterior spinal artery. Both of these arteries are formed

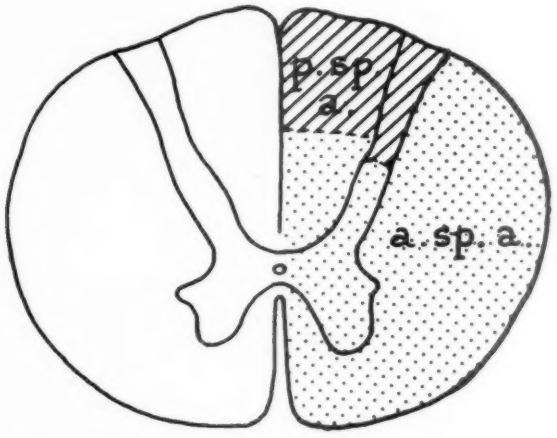

Fig. 4.—Cross-section of spinal cord showing area supplied by anterior spinal artery (*a. sp. a.*) and small segment of posterior horn and posterior column supplied by the posterior spinal artery (*p. sp. a.*).

by the anastomosis of radicular arteries dividing as they reach the cord into an ascending and descending branch. In the cervical region the vascular supply is enriched by the vertebral arteries and the posterior inferior cerebellar arteries, as well as the segmental branches.

In the thoracic and lumbar regions, on the other hand, the radicular branches contributing to the anterior spinal arteries are few and far between, and as a

consequence there is a precarious reserve blood supply of the cord. Less than one-fourth of all nerve roots are accompanied by arterial branches and most of these are in the cervical and upper thoracic region. Of the 6 or 8 branches contributing to the anterior spinal artery, 3 to 5 may be in the cervical and upper thoracic region. Between C 8 and T 9 there are usually 2 unilateral, inconsistently located, small branches to supply this long segment of cord. The largest and most important arterial branch to the cord usually arises somewhere between T 11 and L 3 (usually at L 2). Another small branch may enter below this point (Fig. 5).

The posterior spinal artery has no segmental blood supply of its own inasmuch as it too arises from the anterior spinal artery caudad to the lower cervical region.

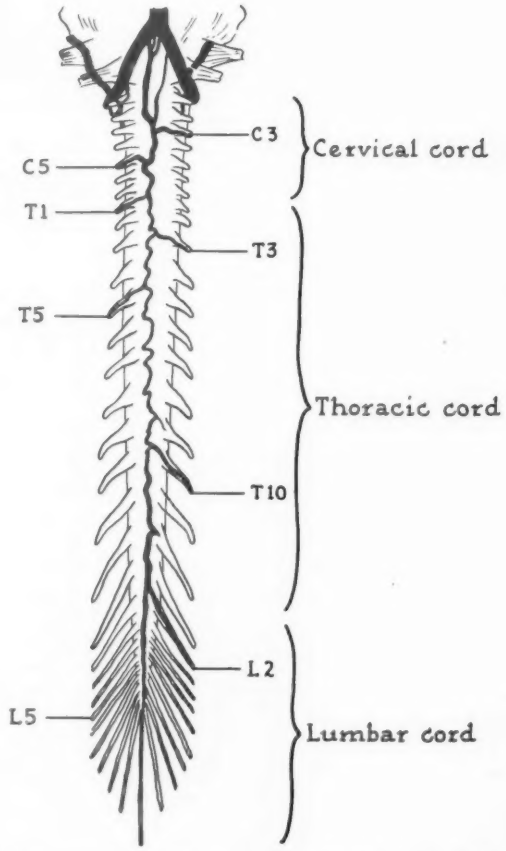

Fig. 5.—Diagram showing segmental blood supply of spinal cord (after Suh and Alexander[28]).

In the various studies of spinal-cord vascularization,[6, 22, 28, 29] all observers have commented upon the wide variation in the location of the vital segmental branches which form the anterior spinal artery and upon which the viability of the cord depends. Although their exact location, size, and the side upon which they may enter is extremely variable, it is abundantly clear that the blood supply of the lower thoracic and lumbar cord is precarious. Neurologists have recognized that a number of inflammatory and degenerative cord lesions are fre-

quently found in the long stretches of thoracic spinal cord midway between nourishing segmental branches.[1] When one or more of these segmental branches is sacrificed in the surgical mobilization of the aorta it is no wonder, therefore, that irreversible cord damage may ensue.

These anatomic studies emphasize the vulnerability of the lower thoracic and upper lumbar cord to aortic cross-clamping and unhappily indicate that no single segmental branch, if preserved, can be counted upon to maintain an adequate arterial supply to the cord.

2. *The Nature of the Resected Aortic Lesion.*—At the present time aortic resection may be indicated for coarctation, atheromatous disease, because of tumor involvement, or because of fistula or aneurysmal dilatation. Data on ischemic sequelae occurring following aortic cross-clamping because of coarctation or obliterative disease are in no way comparable to the other conditions where no period of gradual occlusion has stimulated the development of arterial collaterals. The vast clinical experience[2, 4, 12, 25] in the repair of coarctations of the aorta is, therefore of little importance in gauging safety periods of aortic occlusion for nonobstructive aortic lesions.

3. *The Level of Occlusion.*—Aortic cross-clamping proximal to the innominate artery is limited by cardiac failure or by cerebral ischemia. Clamping immediately proximal to the left subclavian artery is limited by spinal cord damage as it is in the remaining portions of the descending aorta. As the occlusive clamp is moved caudally the period of tolerance to occlusion increases. In human beings without occlusive aortic disease the safe period of aortic occlusion at or just above the left subclavian is normally 52 to 60 minutes,[10, 11, 13, 15, 18] while we, as well as others, have found that periods of 90 to 120 minutes of occlusion are tolerated in the abdominal aorta[18, 20]

4. *The Number of Severed Segmental Arteries.*—Since the anterior spinal artery arises from the occasional intercostal artery it must be remembered that the number and level of the segmental branches of the aorta that are interrupted also may be of significance in producing cord damage. The surgeon usually is forced to ligate 1 or 2 sets of segmental arteries along each end of the severed aorta in order to obtain a cuff sufficiently mobile to permit a satisfactory anastomosis. It is, unfortunately, impossible to determine which of these branches might be critical to the vascular supply of the cord.

5. *Procedures Designed to Minimize Spinal-Cord Ischemia.*—The dangers of spinal-cord damage following cross-clamping of the aorta have been well recognized by vascular surgeons, and two main attempts have been made to minimize this threat.

Temporary vascular shunts of various design have been employed to by-pass the segment of aorta undergoing resection.[8, 23, 24, 27] These shunts have been given experimental but relatively little clinical trial. Their use is not without danger.[23]

Since generalized hypothermia anesthesia diminishes the oxygen requirements of tissues, this technique has been employed to minimize the danger of cord damage during aortic occlusion.[25, 26] It has been shown experimentally to

offer a very substantial protection to the cord, allowing longer periods of aortic interruption with less threat of spinal-cord injury. Despite this protection it is clear from the case here cited that the answer to this problem is not as yet at hand and that even relatively short periods of occlusion of certain portions of the thoracic aorta may on occasion produce severe spinal-cord damage.

SUMMARY AND CONCLUSIONS

1. Factors affecting spinal-cord damage during aortic occlusion are discussed.

2. The precarious segmental nature of the arterial supply to the lower thoracic cord makes it particularly vulnerable to ischemic damage when lower thoracic and upper lumbar segmental vessels from the aorta are interrupted during aortic resection.

3. A case of permanent spinal-cord damage following resection of a lower thoracic aneurysm under hypothermia is presented. The aorta was clamped at the level of T 6 for 63 minutes.

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SUCCESSFUL RESECTION OF A LARGE ANEURYSM OF THE UPPER ABDOMINAL AORTA AND REPLACEMENT WITH HOMOGRAFT

SAMUEL N. ETHEREDGE, M.D.,* JAMES YEE, M.D.,** JOHN V. SMITH, M.D.,** STANLEY SCHONBERGER, M.D.,** AND MERVIN J. GOLDMAN, M.D.,***
OAKLAND, CALIF.

(From the Veterans Administration Hospital)

IN THE past few years great strides have been made in the field of vascular surgery with the introduction of arterial homografts and various plastic prostheses as replacement for resected segments of diseased major vessels. Hufnagel,[1] Gross,[2, 3] Bahnson,[4] Deterling,[5, 6] Brown, Swan,[7] DeBakey and Cooley,[8, 9] and many others[11-14] have reported very encouraging results, particularly in the treatment of aneurysms. The practice of resection and replacement by graft would now seem so well established as the treatment of choice that the previous types of therapy, such as ligation, wiring, wrapping, or endoaneurysmorrhaphy, will either be rapidly discarded or reserved for the particular cases where such a major procedure cannot be carried out for one reason or another.

Schafer and Hardin,[10] writing on the use of temporary polyethylene shunts during the resection of an aneurysm, stated that one of their cases failed because of damage to the diseased aorta above their graft and "that to have been successful our resection and graft replacement should have included the entire abdominal aorta with its celiac, superior mesenteric, and renal branches —a *feasible procedure.*" It was this statement which helped give us the courage to operate upon the patient we are reporting. From a thorough review of the literature, we believe this to be the first such case successfully performed.

CASE REPORT

A 37-year-old Negro male construction worker was hospitalized at the Veterans Administration Hospital, Oakland, California, on July 7, 1954, complaining of recurrent attacks of right chest pain of four days' duration. On July 4, without any exertion or trauma, the patient had experienced a sharp, breath-taking pain in the lower anterior portion of the right side of the chest, resulting in loss of consciousness of approximately ten minutes' duration. At an emergency hospital the patient was told that he had gall bladder trouble and was treated accordingly. There were no respiratory symptoms aside from moderate difficulty in breathing. The chest pain subsided after two hours' time. On the morning of July 5, while dressing, severe right lower chest pain recurred and the patient "blacked out." Moderate pain persisted for six hours. On July 6, the patient was relatively asymptomatic and returned to work. While at work on the morning of July 7, the patient suffered his third attack of

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*Consultant, Peripheral Vascular Diseases, 411 30th Street, Oakland, Calif.
**From the Surgical Service.
***From the Medical Service.

right lower anterior chest pain without any coma on this occasion. The pain subsided after approximately six hours. There were no associated respiratory nor gastrointestinal symptoms.

Past history revealed that in the fall of 1947, while in service on the Philippine Islands, the patient developed fever and left costovertebral angle pain without associated urinary symptoms. The patient suffered three episodes of this nature for which he was hospitalized on each occasion. He was returned to the States and at an Army hospital, in March of 1948, a stone was removed from the left kidney pelvis. Since then the patient was never free from a mild left lumbar pain which, however, was unassociated with fever or urinary symptoms. The patient had gonorrheal urethritis in 1946, but there was no history of a luetic infection.

Physical examination on July 7, 1954, revealed a well-developed, well-nourished ambulatory Negro man in no acute distress, weighing 185 pounds, 5′8″ tall, with a temperature of 99° F., pulse of 68, respirations of 16, blood pressure 120/78. The pupils were round, regular, equal, and reacted to light and accommodation. The fundi showed no arteriosclerotic changes. Vision was good. The nasal septum was intact. The lungs were clear. The heart was not enlarged to percussion. The heart sounds were regular, of good quality, with no murmurs. The peripheral arteries were not palpably sclerotic. The abdomen revealed a well-healed left kidney incisional scar. In the left upper quadrant of the abdomen was a round, firm, fixed, slightly tender mass which had an expansile pulsation and a bruit. There was no abdominal wall rigidity, spasm, or tenderness, and no costovertebral angle spasm or tenderness. Peristalsis was normal. There was no deformity of the back and no tenderness. There were no neurological abnormalities.

Laboratory and X-ray Findings.—Hematology on entry revealed red blood count 3.8 M; hemoglobin 10.9 grams; white blood cells 5,600 with a normal differential count; sedimentation rate of 55 mm. per hour. Urinalysis revealed a specific gravity of 1.029 with a slight trace of albumin on entry which was not present on subsequent urinalyses. Cardiolipin microflocculation test was negative. Blood urea nitrogen was 10 mg. per cent; phenolsulfonphthalein showed 80 per cent excretion of dye in 60 minutes. The right kidney excreted 15 per cent of dye, the left kidney 20 per cent in 15 minutes. Blood cholesterol was 218 mg. per cent total with 91 mg. per cent free. Repeated blood counts were essentially unchanged. X-ray examination of the chest was negative except for questionable slight enlargement of the heart. Excretory urograms were interpreted as showing probable cystic disease of the left kidney. Roentgenogram of the spine showed very marked irregular erosion of the anterior surfaces of T_{12} and L_1 vertebral bodies with perfect preservation of the intervertebral discs, typical of pressure erosion. An aneurysm was suspected. The aneurysmal sac could not be demonstrated on the soft tissue films. An upper gastrointestinal series was negative.

Course and Treatment.—Following admission to the Urological Service, the patient had no recurrence of his right lower anterior chest pain except on the third day of hospitalization, when he had mild pain which lasted for only 45 minutes. However, throughout his course of hospitalization, prior to surgery, the patient experienced mild left flank pain which he had had since 1947. The palpable left upper quadrant mass was suspected of being an aneurysm and the patient was transferred to the Medical Service. An electrocardiogram on Aug. 5, 1954, was within normal limits.

To better delineate the nature of the aneurysm, percutaneous transthoracic aortography employing 70 per cent Diodrast was performed on Aug. 26, 1954, with films taken in oblique views. The lower thoracic and abdominal aorta were well visualized, demonstrating an aneurysm of the upper abdominal aorta, fusiform in type, involving the entire lumen of the aorta. The celiac axis was shown to arise in the middle of the aneurysm. The lower abdominal aorta appeared quite normal. Excellent visualization of the renal circulation as well as pyelograms demonstrating normal kidney pelves were obtained.

In spite of the location of the aneurysm involving the celiac axis and superior mesenteric artery, it was felt that the patient's recent episodes of pain, coma, and anemia were due to hemorrhage from the aneurysmal sac and that lethal rupture of the aneurysm

was imminent. Surgical intervention was deemed indicated and the patient was transferred to the Surgical Service.

Operation.—The patient was taken to surgery on Sept. 20, 1954. He was anesthetized with Pentothal-curare and turned well on his right side. An incision (Fig. 1) was made directly over the course of the eighth intercostal space. The incision was extended into the left rectus sheath and then downward opposite the umbilicus. This combined thoracoabdominal approach afforded a very satisfactory exposure for this large lesion. Exploration was carried out before the diaphragm was completely split in an effort to obtain information about the operability of the lesion. It was noted that the aneurysm was a very large, irregular mass about the size of an adult head (Fig. 1).

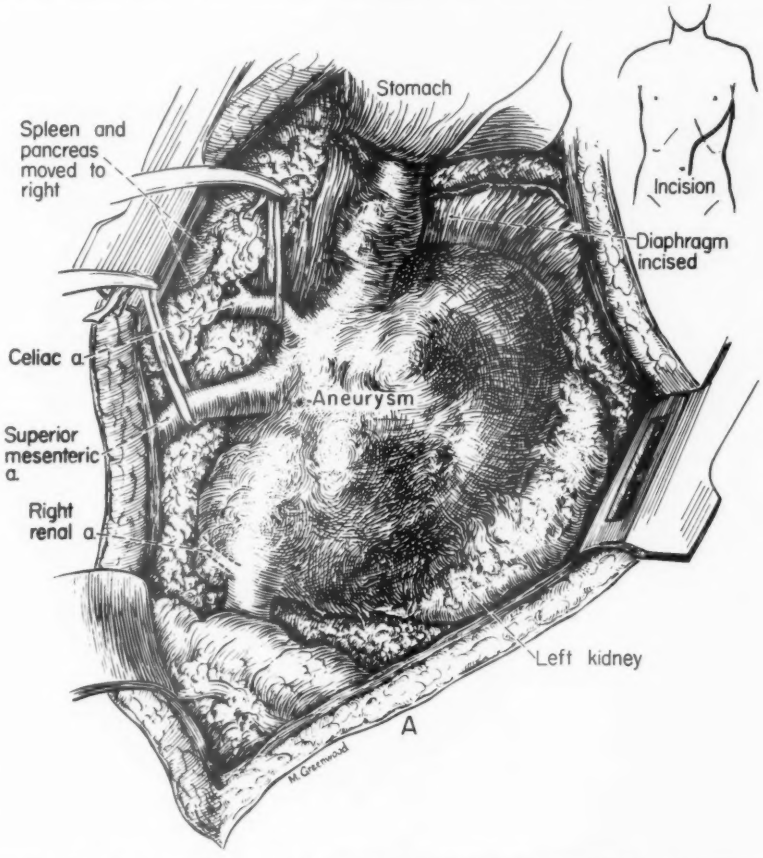

Fig. 1.—*A*, Drawing of the mass in place. Note the secondary aneurysmal dilatation above the large aneurysm; the large size of the superior mesenteric artery as compared to the celiac; and the dense adherence of the left kidney.

The mass had numerous nodulations, some of which were quite soft, indicating marked weakness of the wall in this area. The mass extended out to the lateral abdominal wall on the left side and to well beyond the midline on the right. Superiorly, the mass pushed the diaphragm well up and was very densely adherent to the diaphragm, particularly posteriorly. Inferiorly, the mass extended well below the pole of the left kidney, which was, incidentally, extremely adherent to the mass, and its margins could not be identified.

No contraindication to removal of the mass could be made out by this primary exploration and we decided to proceed carefully with attempt at mobilization of the mass. The aorta was first approached in the thoracic cavity approximately two to three inches above the diaphragm where dissection was carried around the aorta and a heavy rubber tube inserted for control in case of accidental entry into the aneurysmal sac. The left lateral peritoneum was then dissected by sharp dissection and reflected toward the midline.

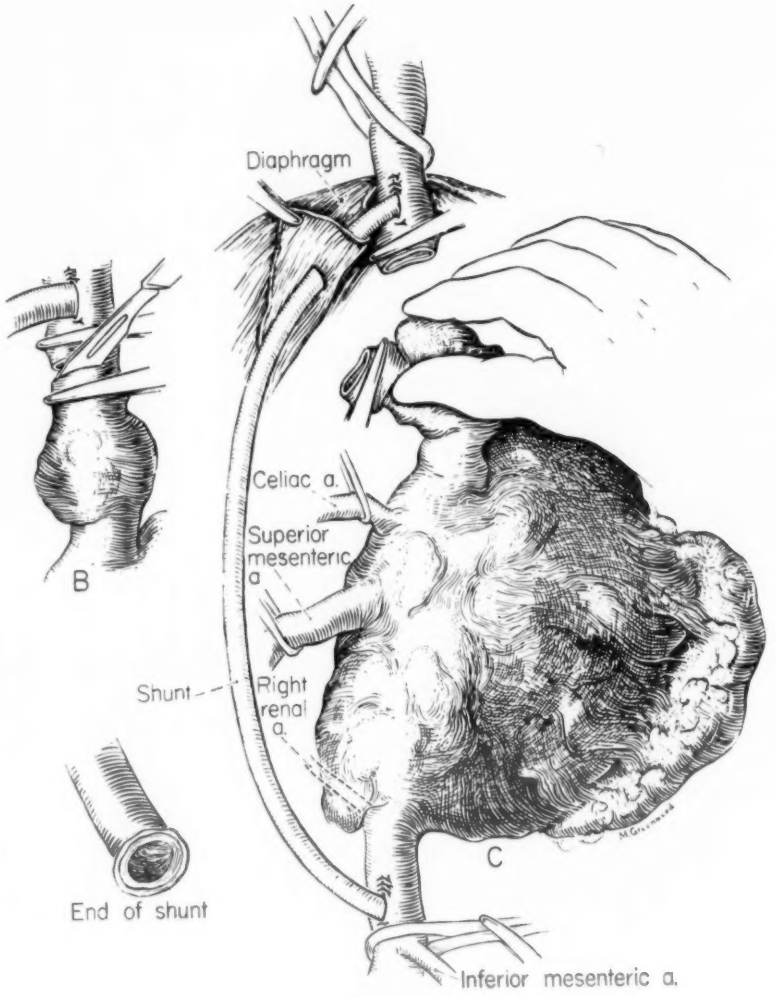

Fig. 2.

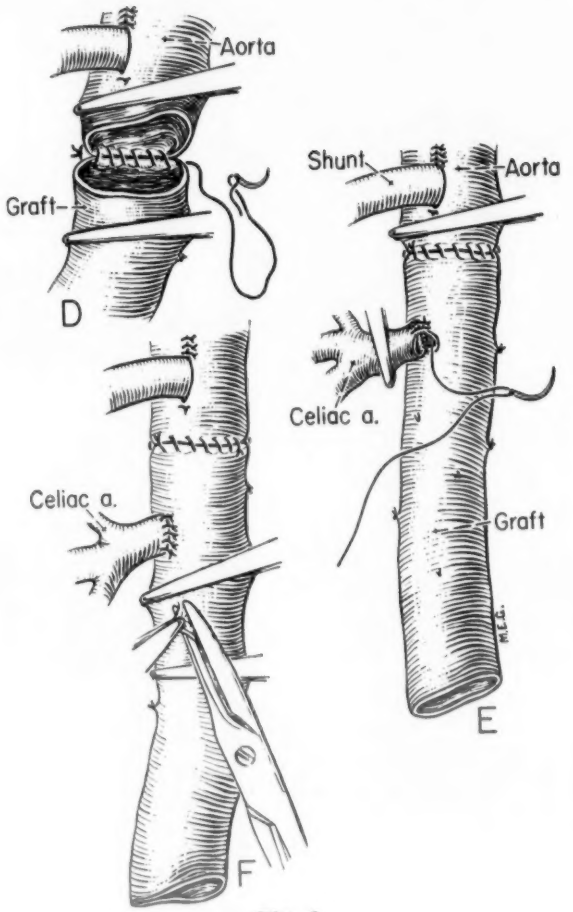

Fig. 3.

Fig. 2.—*B*, Division of the aorta just above the secondary dilatation. Note the shunt already sutured in place above. *C*, Mobilization of the upper portion of the aneurysmal mass. Blood is still flowing into the sac via the shunt.

Fig. 3.—*D*, Anastomosis of the homograft to the upper aorta. A simple over-and-over suture is placed posteriorly. *E*, The aortic anastomosis is completed. The celiac axis is being sutured to a small opening in the graft. *F*, The celiac anastomosis has been completed. The controlling clamp has been shifted to just below the celiac artery. An opening is being made for the superior mesenteric artery.

The spleen was lifted up along with the splenic flexure of the colon, pancreas, and the left kidney, and all of these structures were reflected over together toward the midline. The aneurysm was very densely adherent to the diaphragm posteriorly, and as the mass was reflected the diaphragm was completely separated from its posterior attachments. Several attempts were made to free the left kidney from the mass but all of these only met with failure, either due to the extremely dense adhesions or to the marked vascularity of these adhesions which probably represented some of the renal vessels. These attempts were eventually abandoned as it was felt that if the aneurysm were to be removed the left kidney would have to be removed along with it.

The spleen and its pedicle freed up quite readily and it was possible to completely mobilize the spleen and pancreas and reflect them over into the right subphrenic space where they were left so that the splenic artery could be saved in case it was needed for shunting. As the mass was freed up superiorly, it was noted that there was a distinct bulge of the aorta from just at the level of the diaphragmatic hiatus to a point about 2 cm. above the major aneurysm. It was felt that this represented a small aneurysmal dilatation. This was, however, minute in comparison to the huge sac. Nevertheless, this did further complicate the procedure as we felt that it was absolutely essential to remove this second sacculation if the patient was to profit by the removal of the huge mass. Toward the right side, the dissection was complicated by the necessity of preserving the superior mesenteric and celiac vessels. By very long and tedious dissection, these vessels were eventually identified and dissected free and very carefully preserved.

Posteriorly on the right side, the aneurysmal sac penetrated deeply very far and invaded the right lumbar musculature (Fig. 5). In this area there was considerable acute inflammatory reaction still present and it was felt that this probably accounted for the episodes of acute right upper quadrant pain which had been the cause of the patient's original hospitalization. The right renal artery could be palpated but was never well visualized due to the fact that it was overlaid by the huge mass. Inferiorly, the aorta seemed to be in relatively good condition and its walls not too thick except posteriorly where rather marked plaque formation could be made out. It was our hope that the aneurysm could be completely mobilized for its entire circumference before thinking of division of the aorta, but this became very obviously impossible as we proceeded posteriorly where the aneurysm had markedly eroded at least two of the vertebrae, thought to be T 12 and L 1. Complete mobilization then had to be deferred until we were ready to proceed with the actual excision of the aneurysm.

It was felt that a form of shunting past the involved area would be absolutely necessary in view of the long procedure contemplated. Therefore, following the procedure advocated by Schafer,[10] a polyethylene shunt, measuring 5 mm. inside diameter, 7 mm. outside diameter (Fig. 6), was inserted into the aorta at the superior margin of dissection after control using Pott's clamps had been applied above and below the level (Fig. 2, *B*). A short longitudinal incision was made in the anterior wall of the aorta. The end of the tube with its small flare was inserted into the aorta and sutured in place with several interrupted No. 5-0 Deknatel arterial silk sutures. The controlling clamps on the aorta were then immediately removed. The lower end of this polyethylene tube was then inserted into the lower abdominal aorta just above the bifurcation in a similar fashion.

At this point a very distressing complication arose. It was observed that the plaque on the posterior abdominal aorta, which was noted on palpation, was much thicker than had been anticipated. In an attempt to remove a portion of this plaque, a small rent was inadvertently made in the posterior wall of the aorta which caused considerable difficulty in its repair. However, this repair was accomplished, although we were concerned about some reduction in the size of the aorta at this level.

The polyethylene shunt had been passed through a small hole in the diaphragm which took the shunt well out of the field of operation where it was very unlikely to be dislodged. A rubber tube control was placed around the distal aorta but above the level of the entrance of the shunt, and this control was laid loosely in place and not tightened down. A site for division of the aorta superiorly was chosen just above the old site of the diaphragmatic hiatus. The aorta was divided at this level between coarctation clamps and was dissected

controlling clamp has been shifted to just below the celiac artery. An opening is being made for the superior mesenteric artery.

down as far as possible (Fig. 2, *C*). The arterial homograft, which consisted of the aorta of a young adult male, was then sutured to the upper end of the aorta using a continuous arterial silk suture (No. 4-0 Deknatel) (Fig. 3, *D*).

The graft was then laid in place along the large aneurysm, with a little tension placed upon it to approximate its future position after the mass had been removed. At the estimated level of the celiac artery a small ellipse was removed from the aortic graft wall. The patient's celiac artery was divided between small arterial clamps, and an anastomosis of the celiac artery to the graft was carried out (Fig. 3, *E*). This anastomosis was extremely difficult and the most trying portion of the entire operation. The patient's celiac artery was extremely friable, being more so than a vena cava or portal vein ordinarily is. The vessel tore several times before the anastomosis could be completed. Once anastomosis had been completed, the controlling clamp was placed just distal to the anastomosis and the controls were removed from the aorta above, permitting blood to flow down through the aorta and

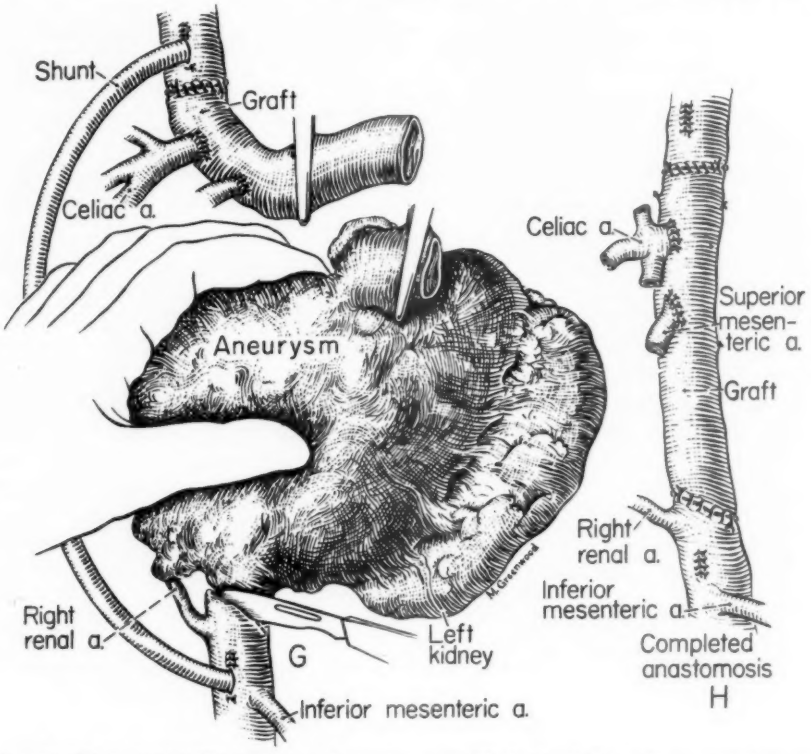

Fig. 4.—*G*, The controlling clamp has been shifted to below the superior mesenteric. The entire mass is being mobilized. The right renal artery has finally been visualized and the aorta is being divided on a bias just above this vessel. A controlling clamp was in place just below the renal artery at this time but is not shown. *H*, The graft is completely sutured in place. The shunt and all controlling clamps have been removed.

into the celiac artery. An identical procedure was then carried out anastomosing the patient's distal superior mesenteric artery to the homograft (Fig. 3, *F*). As soon as this anastomosis had been completed, the control on the graft was moved just distal to the superior mesenteric anastomosis and blood was permitted to flow into both celiac and superior mesenteric arteries. It was estimated that approximately twelve to fifteen minutes was required for each anastomosis, during which time the blood flow was shut off to both of these major vessels.

At this point we prepared for very rapid action. The distal aorta was clamped, and immediately the large aneurysm was dissected free as fast as was possible. All of the intercostal and lumbar vessels that could be identified were divided, clamped, and ligated. The large sac was dissected from the vertebral column where it eroded the two vertebral bodies and also from the right lumbar musculature. Once the mass could be lifted up out of the

abdominal cavity it was possible to observe the right renal artery and to divide the aorta slightly on the bias and preserve an estimated 1 cm. of reasonably healthy aorta above the right renal take-off. The lower end of the graft was then quickly sutured to the distal aorta (Fig. 4, *G*).

As soon as the anastomosis was completed the control below the superior mesenteric was removed, the inferior control removed, and blood was allowed to flow completely through the graft. A very satisfactory pulsation of all the vessels was immediately detected. All small bleeding points, both in the anastomotic line and in the area of dissection, were carefully searched for and controlled. Several pieces of Gelfoam were placed down in the defect in the vertebral bodies. The spleen which, as previously stated, had been preserved up to this time for possible emergency use was now removed. The polyethylene shunt was next removed between controls (Fig. 4, *H*).

The posterior peritoneum was closed where possible, but this was quite incomplete due to the large defect created. The diaphragm was reconstructed and reconstituted as far as was possible. A right-angle chest tube was inserted and brought out through the ninth intercostal space. The patient showed some signs of unresponsive shock near the end of the opera-

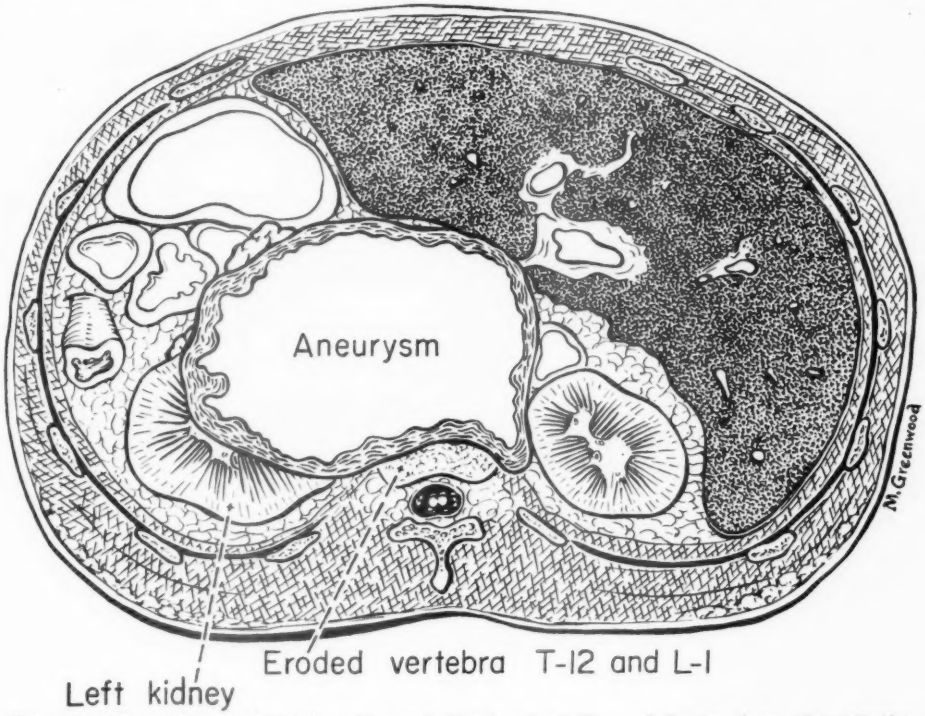

Fig. 5.—Schematic cross-section through the level of T_{12} and L_1 to show the relative size of the aneurysm, its adherence to the left kidney, the erosion of the vertebral bodies, and the deep dissection in the right paravertebral space.

tion, and at that time he was started on intravenous hydrocortisone. He had been receiving continuous drip of Levophed for some period at that time. The patient's general condition at the close of the operation was reasonably satisfactory considering the extremely prolonged procedure.

Pathology Report.—Gross: The specimen consisted of an irregular mass measuring 11 by 10 by 8 cm. At one point a segment of aorta could be seen. On opening this it was markedly distorted; however, a large aneurysm filled with friable clotted blood was found attached to the aorta. The surface of the aorta showed numerous bright-yellow atherosclerotic plaques. There was no evidence of syphilis or of other arteriole disease. A large mass of skeletal muscle was also present, associated with the aneurysm. Sections of the aneurysm wall showed at one point aorta with considerable subintimal fibrosis. The elastic tissue was

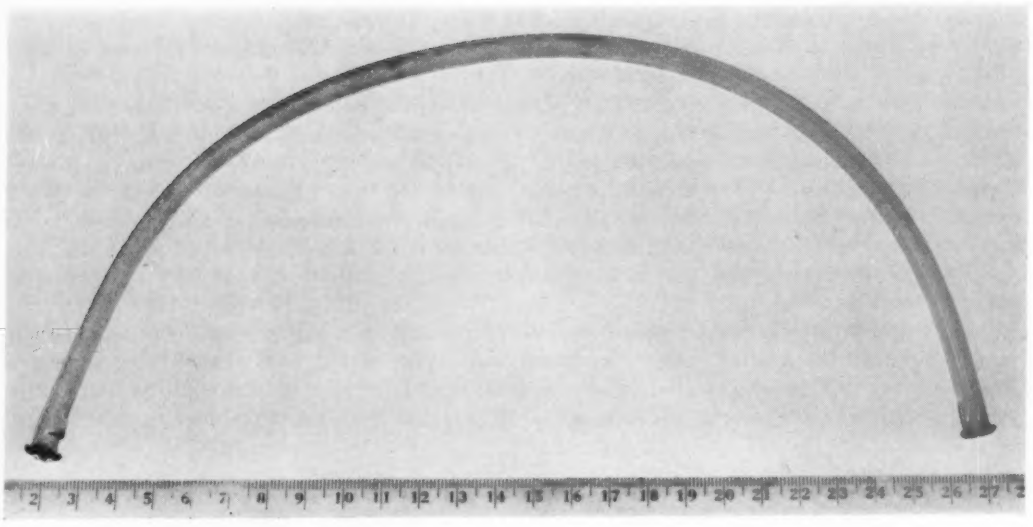

Fig. 6.—The polyethylene shunt used.

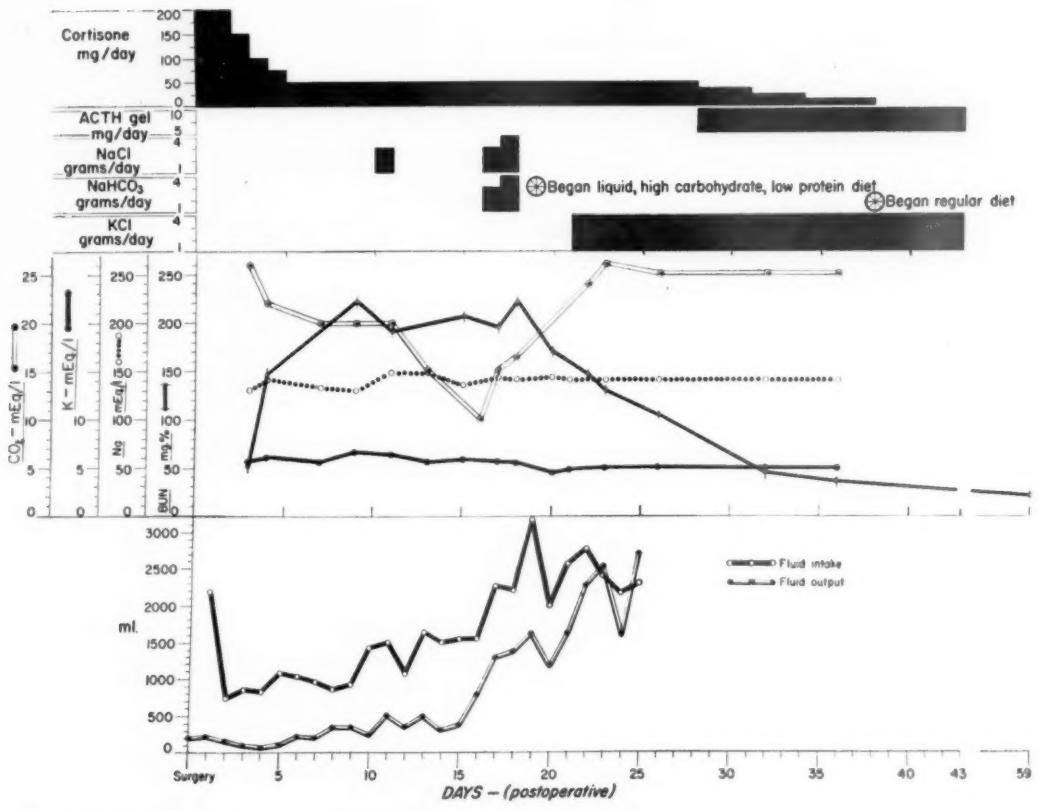

Fig. 7.—Graph showing management of the postoperative complication of urinary suppression. The hormones and electrolytes given are shown at the top of the graph, the blood chemistries are illustrated in the middle section, and the fluid balance graph is shown below.

irregular, broken in a few areas but, in general, was mainly intact. At the point where the aneurysm was formed the media had been replaced by fibrous tissue and the wall of the aneurysm was composed only of collagenous connective tissues. A few sites of lymphocytic infiltrations were present. The wall of the aneurysm was covered by clotted blood. *Diagnosis:* Aortic aneurysm; atherosclerosis.

Postoperatively, the peripheral circulation was adequate, the posterior tibial pulsations being palpable at all times. There was no paraplegia or paresis of the lower extremities and no other evidence of spinal cord ischemia. The postoperative course was uncomplicated except for the development of acute tubular necrosis. This complication was followed very closely and successfully* (Graph, Fig. 7).

A regimen was then started of restricted fluid intake, calculating the 24-hour intake on the basis of 750 c.c. plus the amount of urine excreted in the previous 24-hour period. To this total amount of water was added 300 Gm. of glucose daily. The fluid intake and output and serum electrolytes are illustrated in Fig. 7. The patient received no additional electrolytes except as illustrated in the chart. At no time did the patient develop any sign of pulmonary or peripheral edema. The blood pressure remained within normal levels throughout. The serum potassium never rose above 6.5 meq. per liter. Serial electrocardiograms were all within normal limits. Diuresis (urine volume over 500 c.c.) began on the sixteenth postoperative day. No problems were experienced in fluid and electrolyte balance during the diuretic phase.

Because of the unknown status of the patient's own adrenals, with the known sacrifice of the left adrenal gland, he received 100 mg. of hydrocortisone intravenously and 100 mg. of cortisone intramuscularly during surgery. In spite of the development of the acute renal insufficiency, we felt obligated to continue hormonal therapy (Fig. 7). Supplementary potassium was not added until the patient had reached the diuretic phase. The blood urea which had reached a high of 235 mg. per cent on the sixteenth postoperative day gradually came down to 19.3 mg. per cent by Dec. 3, 1954. The patient was discharged from the hospital on Jan. 13, 1955. He has been followed at regular intervals since that time, and continues in very satisfactory condition.

COMMENT

There were numerous outstanding points of interest which arose during the management of this tremendous problem. We will discuss some of these at random.

1. The type of aneurysm in a patient of this age. As noted, the patient was only 37 years of age and had had previous left renal surgery. We therefore considered preoperatively the outside possibility of the aneurysm being traumatic in origin. There was absolutely nothing in the history or serology to suggest a syphilitic origin. The gross findings at surgery and the later pathologic study proved the definite arteriosclerotic nature of the aneurysm.

2. The extensive involvement of the great vessels of the upper abdomen naturally greatly complicated and prolonged the dissection as each individual organ and vessel had to be preserved until the decision could finally be reached that resection was possible. The difficulty in identifying the renal vessels was particularly disturbing, and this was apparently due to the invasion of the left renal pedicle by the aneurysm and the posterior rotation of the right renal vessels due to the huge aneurysmal sac overriding the aorta at this level.

3. The secondary dilatation of the aorta between the aneurysm and the diaphragm would not seem to offer much of a problem, but we felt that this diseased area should definitely be included in the resection and this additional

*By Dr. Mervin J. Goldman of the Medical Service.

segment moved the dissection for control of the thoracic aorta and insertion of the shunt just high enough out of the operative field to make these procedures unpleasantly difficult and painstaking.

4. The marked posterior involvement of the distal aorta which resulted in our posterior leak at that level, threatened for a while to nullify all of our efforts. We had been impressed by the pliability of the anterior wall and were somewhat taken by surprise by the extensive arteriosclerotic involvement posteriorly. We have been impressed by this predominant posterior involvement of the aorta, iliacs, and femorals on numerous previous cases and can think of no satisfactory explanation, nor have we encountered any such explanation in the literature.

5. The use of polyethylene shunts has been well described by Schafer and Hardin[10] and others. Although the shunt we used measured only 0.5 cm. in inside diameter, we feel that it undoubtedly played a vital role in the handling of this case. By its use and by the step-procedure which we devised for division of the great abdominal vessels one at a time, we were able to keep blood flowing into all of the vital organs except for relatively short periods of time. We are sure this contributed to the patient's excellent postoperative course, which, except for the oliguria, was amazingly smooth. (On the third postoperative day he requested to be moved from the room with the sick people in it.)

6. It is impossible to state at this time that the homograft is the ideal substitute for the resected vessel since the literature is beginning to report excellent results with some of the plastics which would be much simpler to obtain and to handle. We have had no experience with such materials. Our own graft was obtained and prepared by our local artery bank. We are at present using the quick-freeze method of preparation, but thanks to a grant from the Heart Association will eventually change to the freeze-dry process.

SUMMARY

A case is reported of what we believe to be the first successful resection of an arteriosclerotic aneurysm of the upper abdominal aorta with division and anastomosis of the celiac and superior mesenteric arteries to the homograft replacement.

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ADDENDUM

The patient has been followed at regular intervals and was last seen on Oct. 14, 1955. He has regained his preoperative weight and strength and has returned to active construction type of work.

CHOLECYSTOGRAPHIC STUDIES DURING AND IMMEDIATELY FOLLOWING ACUTE PANCREATITIS

VAN G. KADEN, M.D., JOHN M. HOWARD, M.D., AND LEONARD C. DOUBLEDAY, M.D., HOUSTON, TEX.

(From the Departments of Surgery and Radiology, Baylor University College of Medicine, and the Jefferson Davis Hospital)

FAILURE of gall-bladder visualization has been considered roentgenographic evidence, in most patients, of cholelithiasis. This failure of visualization has been attributed to nonfilling of the gall bladder (cystic duct obstruction) or to the loss of the absorptive (concentrating) function by the damaged gall bladder. Because of the essential difference in clinical management of patients with acute cholecystitis and acute pancreatitis, it is necessary to distinguish accurately between the two diseases. Since acute cholecystitis is associated with roentgenographic nonvisualization of the gall bladder, it appeared important to study patients with acute pancreatitis by cholecystography during the earlier phases of the illness, at which time the differential diagnosis must be made.

This has been a study of 23 patients with acute pancreatitis in whom gallstone disease has subsequently been excluded. Selection of patients was dependent upon the subsidence of symptoms and the return of gastrointestinal function as indicated by clinical signs. Each patient was given the cholecystographic medium (Priodax* or Telepaque†) orally, or Cholografin‡ intravenously, in standard doses, during the first week of hospitalization. Cholecystography was then repeated during the ensuing outpatient visits.

Patients experiencing vomiting or diarrhea during their roentgenographic studies have been excluded.

OBSERVATIONS

Ten normal or chronically ill subjects served as controls. Gall-bladder visualization was normal in nine of the studies, the tenth being unsatisfactory because of the resultant diarrhea.

Of the 23 patients studied during their hospitalization, the gall bladder was clearly visualized in only 8 (Table I). In two additional instances, the concentration of opaque material in the gall bladder was interpreted as subnormal. Follow-up studies of these two patients later demonstrated a normal concentration of dye in the gall bladder. In two other patients a very faint,

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*"Priodax," brand name for Iodoalphonic acid (Schering Corporation), (beta[4-hydroxy-3, 5-diiodophenyl] alpha-phenyl-propionic acid).

†"Telepaque," brand name for Iodopanoic acid (Winthrop-Stearns, Inc.), (3-amino-2,4,5-triiodophenyl)-(2-ethyl propionic acid).

‡"Cholografin," brand name (E. R. Squibb & Sons) for N,N¹-adipyl-bis (3-amino-2,4,6-triiodo)-benzoic acid.

barely discernible shadow which probably represented the gall bladder was present on the initial examination. Subsequent examination of these patients demonstrated normal visualization.

TABLE I. CHOLECYSTOGRAPHY DURING ACUTE PANCREATITIS IN PATIENTS WITH NORMAL BILIARY TRACTS

GALL-BLADDER VISUALIZATION DURING ACUTE PHASE*	NUMBER OF PATIENTS
Normal	8
Impaired visualization	2
Very faint visualization	2
Nonvisualization	11
Total	23

*The first seven days of illness.

Roentgenographic studies of the remaining 11 patients during the period of hospitalization failed to delineate the gall bladder. Eight of these patients, with nonvisualization during the early course of their illness, demonstrated a normal gall bladder outline when restudied during the ensuing months. Three additional patients with nonvisualization were found to have acute pancreatitis and a grossly normal gall bladder at the time of laparotomy. Many other patients demonstrated nonvisualization during their illness with acute pancreatitis and demonstrated normal concentration at a later date, but, because they had gallstones, they are excluded from this series.

The clinical observations are demonstrated by the following three case presentations.

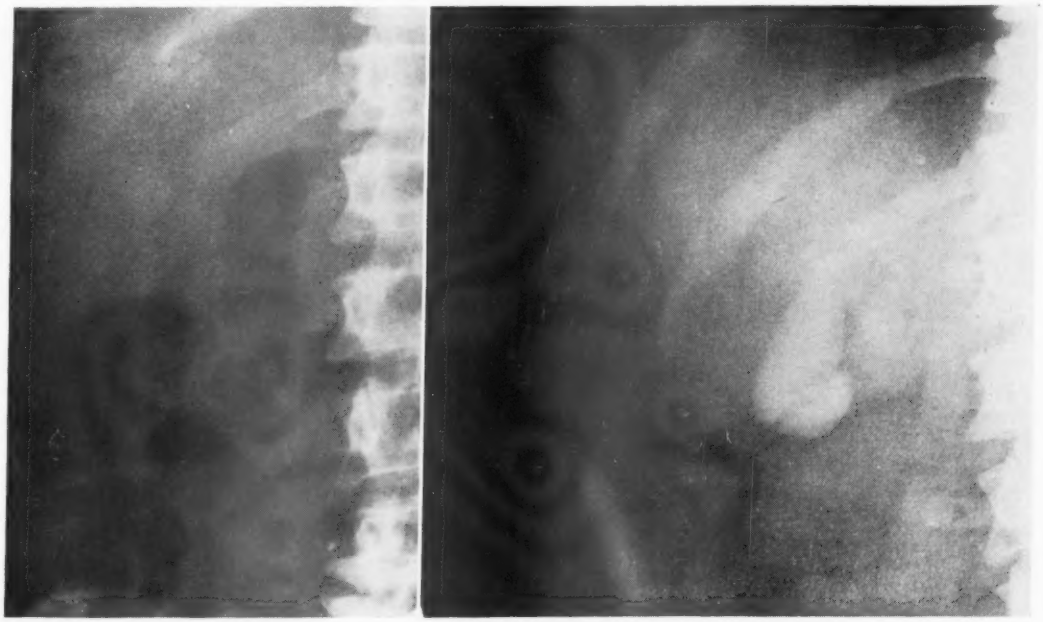

Fig. 1.—Nonvisualization of the gall bladder followed by visualization of a normal gall bladder after recovery from acute pancreatitis.

CASE 1.—A 27-year-old Negro woman was admitted to Jefferson Davis Hospital on May 18, 1951, complaining of nausea, vomiting, and cramping epigastric pain of four days' duration. She had experienced a similar episode two years previously. Further questioning

revealed a history of drinking approximately one-half pint of whisky daily during the preceding two years. There was no history suggestive of gallstone disease. Examination revealed a moderately obese woman in acute distress, with direct and rebound tenderness most marked in the epigastrium. The bowel sounds were diminished. The serum amylase concentration,

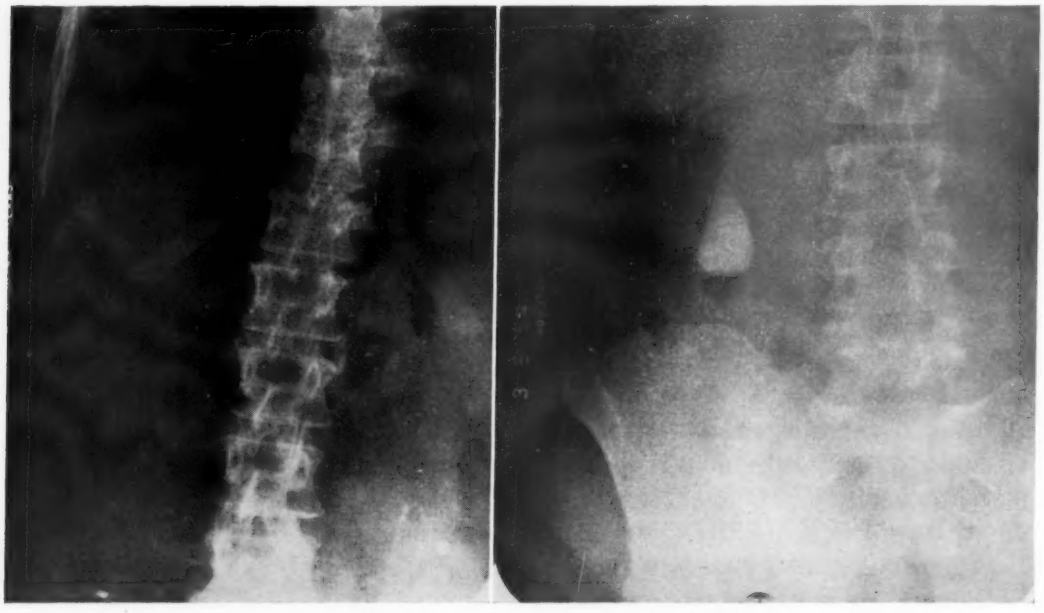

Fig. 2.—Roentgenograms demonstrating the recovery of cholecystic function between the eighth and twelfth day after onset of acute pancreatitis.

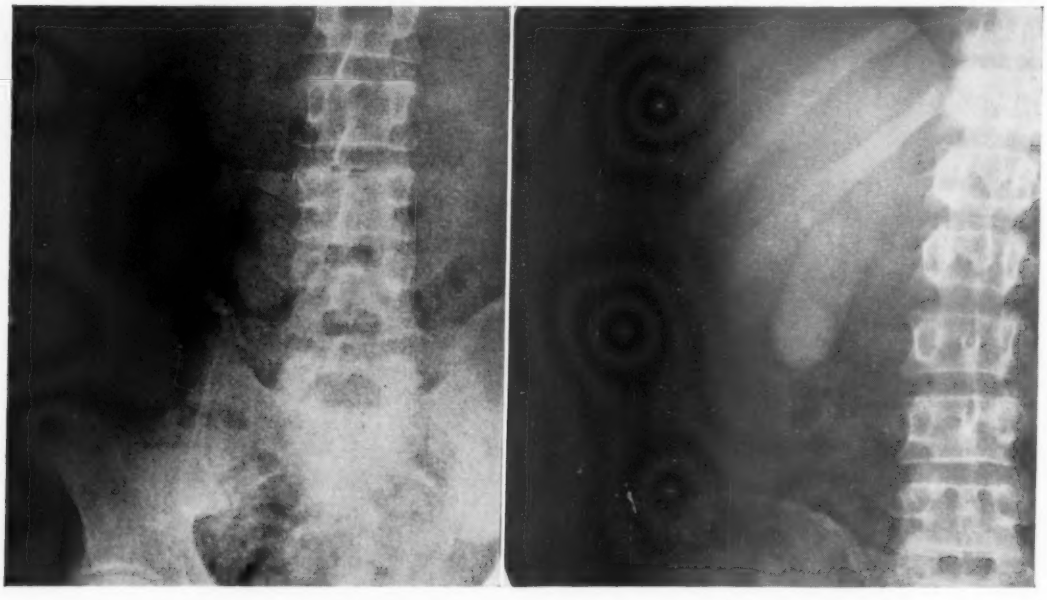

Fig. 3.—Faint visualization on the fifth day of illness followed by normal visualization on the fourteenth day.

elevated on admission, had returned to normal by the third hospital day. Meanwhile, she had become asymptomatic. Cholecystography on the fifth hospital day (ninth day of illness) failed to outline the gall bladder. Five months after the acute illness, cholecystography revealed normal visualization of the gall bladder without evidence of cholelithiasis (Fig. 1).

CASE 2.—A 46-year-old Negro woman was admitted to Jefferson Davis Hospital with a history of nausea, vomiting, and generalized abdominal cramping and distention of four days' duration. Although this represented her first episode of acute abdominal pain, she had a long-standing history of alcoholism with an alcoholic bout preceding this illness. The abdomen was distended and tympanitic with absent bowel sounds. There was generalized rebound tenderness, more marked in the epigastrium. The initial serum amylase concentration on the sixth day of her illness was 76 mg. per cent, the normal being 20 to 40 mg. per cent. This rose to 109 mg. per cent the following day but returned to 60 mg. per cent on the eighth day of her illness. By the eighth day she was tolerating an oral fluid intake. Cholecystography (Priodax) was then attempted but failed to outline her gall bladder. This test was repeated twelve days after onset of the illness and showed satisfactory concentration of dye in the gall bladder without evidence of stones (Fig. 2).

CASE 3.—An 18-year-old white woman was admitted to the hospital on June 28, 1951, with a 12-hour history of cramping epigastric pain radiating straight through to her back and under her right shoulder. There was accompanying nausea and vomiting. She had suffered a similar, less severe episode two years earlier. Examination revealed an obese patient in marked distress with severe direct and rebound tenderness in the epigastrium. Peristaltic sounds were absent. The serum amylase concentration, 116 mg. per cent on admission, subsided to 19 mg. per cent by the fifth hospital day. A cholecystogram on the fifth day showed faint visualization of the gall bladder without stones. A second cholecystogram, 14 days after the onset of the illness, revealed a normal concentration of dye in the gall bladder (Fig. 3).

DISCUSSION

The correct differential diagnosis between acute cholecystitis and acute pancreatitis is essential since one responds well to early surgical intervention, the other to nonoperative management. The distinction may not be an easy one, especially when the patient is first seen several days after the onset of the disease. Nonvisualization of the gall bladder by cholecystographic methods, being characteristic of acute cholecystitis, has been used as a diagnostic distinction.

The results of this study indicate that such a diagnostic criterion is invalid. Nonvisualization occurs frequently, almost characteristically, during the first week of illness with acute pancreatitis. At the time when diagnostic assistance is needed, this study is of value only if visualization of the gall bladder results.

This observation is a confirmation of the study by Silvani and McCorkle[4] who demonstrated that nonvisualization was not infrequent, even though the radiopaque medium was administered intravenously. A similar observation was made by Nothman[3] during the study of patients with chronic relapsing pancreatitis. Failure of gall bladder visualization is apparent in several of the patients reported by Elman.[1] Radakovich and his associates[6] added additional clinical observations demonstrating a reduced opacification of the gall bladder during acute pancreatitis, but in most of their patients with nonvisualization, cholecystic disease was not excluded. They presented experimental evidence that, in dogs, ligation of the pancreatic ducts or pancreatectomy resulted in a decreased absorption of the cholecystographic media from the bowel and a resulting nonvisualization of the gall bladder.[5] After the lapse of a few days, nonvisualization resulted even if the media were given intravenously. They concluded that the pancreatic secretion controlled, in part, not only the absorption of the cholecystographic media from the in-

testinal tract, but also the cholecystic tone or absorption. Studies of battle casualties indicated that nonvisualization often followed severe injury of any type.[2] This would strongly suggest that the phenomenon was not specific for direct pancreatic injury. Since the intravenous administration of the cholecystographic medium in the earlier studies, as well as in the last two patients of this series, also resulted in nonvisualization, failure of intestinal absorption of the medium is not the only defect. Paralytic ileus of the gall bladder with failure of intermittent emptying and refilling might be postulated as the basic underlying defect. Diminution in the absorption of water by the gall bladder may prove to be a factor.

SUMMARY

Cholecystographic studies of 23 patients during the first week of convalescence from acute pancreatitis indicated nonvisualization of the gall bladder in 11 instances and impaired function in an additional 4 instances (total of 65 per cent). Cholecystograms during follow-up studies or surgical exploration indicated that these patients had no organic disease of the gall bladder. The return of cholecystic function, as indicated by cholecystography, characteristically occurred within a month after the symptoms of acute pancreatitis had subsided.

During the first week of illness with acute pancreatitis, a cholecystogram is of diagnostic assistance only if the gall bladder is visualized, for nonvisualization at this time does not necessarily indicate organic cholecystic disease.

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SEGMENTAL ARTERIAL SPASM OF THE BRACHIAL ARTERY

Report of Case Treated by Procaine Into Median Nerve

Jacob Kulowski, M.D., St. Joseph, Mo.

(From Missouri Methodist Hospital)

THE mechanical causes of circulatory disturbances associated with supracondylar fracture of the humerus in children are well known, and feared by laymen and doctors alike. Not so well known, perhaps, is the entity, called by various names, but best described clinically as (reflex) segmental arterial spasm.

CASE REPORT

L. L. A., an 8-year-old white girl, was admitted to the Missouri Methodist Hospital on the night of April 22, 1955, with a compound supracondylar fracture of the right elbow, following a fall from a horse.

On examination there was the usual, obvious deformity associated with this type of injury, and a 2 cm. contused puncture wound in the middle of the antecubital fossa, through which the jagged end of the lower humerus protruded.

Most striking was the loss of the radial pulse at the wrist, with moderate coldness and slight swelling of the fingers and hand. Because of poor cooperation more detailed information with regard to the neurocirculatory situation could not be ascertained.

Fig. 1 shows the roentgenographic appearance of the right elbow on admission to the hospital (about three hours after injury).

Course.—Within an hour débridement and open reduction of the badly displaced fracture surfaces were carried out (about four hours after injury). It was felt that the radial pulse would function as soon as the mechanical pressure upon it was relieved by reduction. The reduction was easily accomplished but the radial artery failed to resume pulsations. After waiting for this to happen without success for several minutes, segmental arterial spasm was suspected, and the antecubital wound was enlarged to afford exposure of the brachial artery and median nerve. The veins and nerve appeared normal; but the brachial artery was attenuated and almost stringlike. The artery was absolutely pulseless and remained so after several minutes of manipulation and massage; despite the fact that there was now no evidence of any kind of pressure upon it or evidence of arterial contusion or laceration.

The close proximity and healthy appearance of the median nerve, on the other hand, suggested, even invited, a trial of injection of procaine into it. Three centimeters of 1 per cent procaine was instilled under the nerve sheath with a fine needle. In a few moments, the brachial artery began to pulsate feebly but surely. Another injection was made, following which the pulsations increased in volume and could soon be palpated also at the wrist. Reduction was maintained with the elbow at about 80 degrees, the wound loosely closed without drainage, and a posterior plaster splint applied.

The immediate and subsequent recovery was uneventful. Fig. 2 shows the lateral roentgenogram of the patient's elbow three weeks after operation. When seen last on June 10, 1955, there was no evidence of neurocirculatory disturbance, and the patient exhibited a 50 per cent range of motion, which was expected to attain normalcy in due course of time.

COMMENT

This case was undoubtedly one of segmental arterial spasm because the vessel had been fully exposed for at least 3 to 4 cm., and exhibited no signs of

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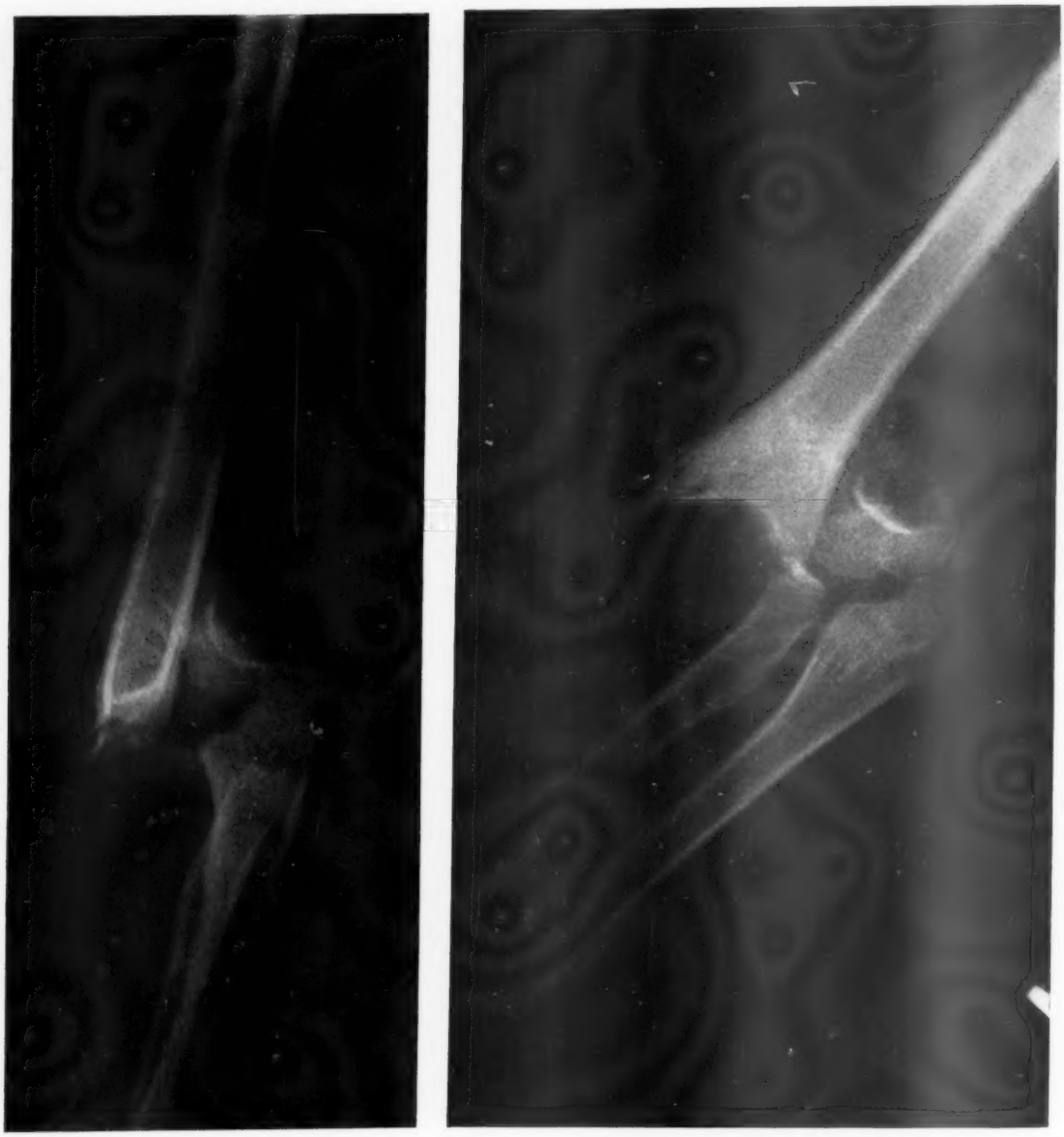
local compression, injury, or thrombosis. The condition and its dramatic response to local procaine injection into the median nerve had to be seen to be fully appreciated, especially on the background of less responsive experiences with similar clinical threats in the past. Since the condition is thought to be due



A. *B.*

Fig. 1.—(April 22, 1955.) Anteroposterior (*A*) and lateral (*B*) roentgenograms of right elbow before operation.

to reflex sympathetic activity, it is suggested that this reflex arc was broken by procaine injected into the median nerve.

Clinicians faced with similar situations, especially when the fracture is already compounded and fresh, ought not to hesitate in exposing the antecubital

structures for a look at the brachial artery (after the fracture has been reduced). In the presence of segmental arterial spasm, injection of procaine into the median nerve should be given a clinical trial, since it is the least complicated and least disturbing locally of all other methods already suggested in the literature.

In so far as I am able to tell, this is the first case of segmental arterial spasm to be so treated successfully.

Fig. 2.—Lateral roentgenogram of same patient's right elbow, three weeks after operative reduction.

SUMMARY

A case of severe supracondylar fracture of the elbow in a child has been reported. The situation was further complicated by absence of the radial pulse and threat of Volkmann's ischemic contracture. The circulatory embarrassment was thought to have been due to mechanical compression of the brachial artery at the site of injury. Instead, there was found at operation a so-called arterial spasm of the brachial artery. The latter was relieved by injection of procaine solution into the median nerve which resulted in full restoration of the circulation in the affected extremity. This method of treatment is considered to be original and is suggested for similar cases of impending ischemia after satisfactory reduction, closed or open, has failed to produce a palpable arterial pulse at the wrist.

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Recent Advances in Surgery

CONDUCTED BY ALFRED BLALOCK, M.D.

THE HEALING AND FATE OF ARTERIAL GRAFTS

LESTER R. SAUVAGE, CAPTAIN, MC, USAR,* AND SIGMUND A. WESOLOWSKI, CAPTAIN, MC, USAR,** WASHINGTON, D. C.

(From the Division of Surgery, Army Medical Service Graduate School, Walter Reed Army Medical Center)

THROUGH the study of experimental arterial grafts we have come to realize that to understand their healing is to understand their fate. We have observed the same pattern of healing in autografts, homografts, heterografts, and Orlon tubes despite the fact that their end results varied widely. This variation is explainable on the basis of whether the cellular viability and fiber continuity of the original graft were maintained or not. If they were lost, the time at which this loss occurred was of the greatest importance in determining the gross morphologic fate of the graft. The objectives of this paper are first to present and comment upon the data of 230 experimental grafts which appear to be transferable to the human patient and second to consider certain aspects of reconstructive arterial surgery in light of these data.

EXPERIMENTAL METHODS AND RESULTS

The material of this paper consists of 230 grafts implanted in 170 animals (153 young pigs and 17 mature dogs). Of these grafts, 222 were tubes (197 arterial, 8 venous, and 17 Orlon) used to span arterial gaps, 3 were segments of the host aorta from which the inner wall had been stripped out, and 5 were Orlon patches used to bridge musculofascial defects.

Sterile precautions were observed in all phases of graft procurement, preparation, and implantation. The operative procedures were performed under Nembutal (intraperitoneal)–ether (endotracheal) anesthesia for the pigs and under Nembutal (intravenous) anesthesia for the dogs.

The grafts implanted in the pigs averaged 6 cm. in length. They were used to bridge defects of the upper descending thoracic aorta which had been made by exising segments averaging 5 cm. in length. All anastomoses were performed with 5-0 silk placed as a continuous end-on coaptation stitch. The

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Upper end of graft is to the right in all figures of gross specimens in this paper.

All photomicrographs of vascular grafts in this paper are of longitudinal sections with the inner surface at the top.

*Present address, Department of Surgery, University of Washington, Seattle, Wash.

**Present address, Department of Surgery, State University of New York, Brooklyn, N. Y.

average time required for graft implantation was fourteen minutes. After measurements were taken, the graft was pleuralized and the chest closed. Anticoagulants were not employed. Antibiotics consisting of 300,000 units of procaine penicillin and 0.5 Gm. of streptomycin were administered intramuscularly to all animals at the completion of surgery, but were not repeated postoperatively for the pigs, while they were administered once daily for the first five postoperative days to all dogs. A few days after operation the pigs were transferred to a farm and kept until they either died spontaneously or

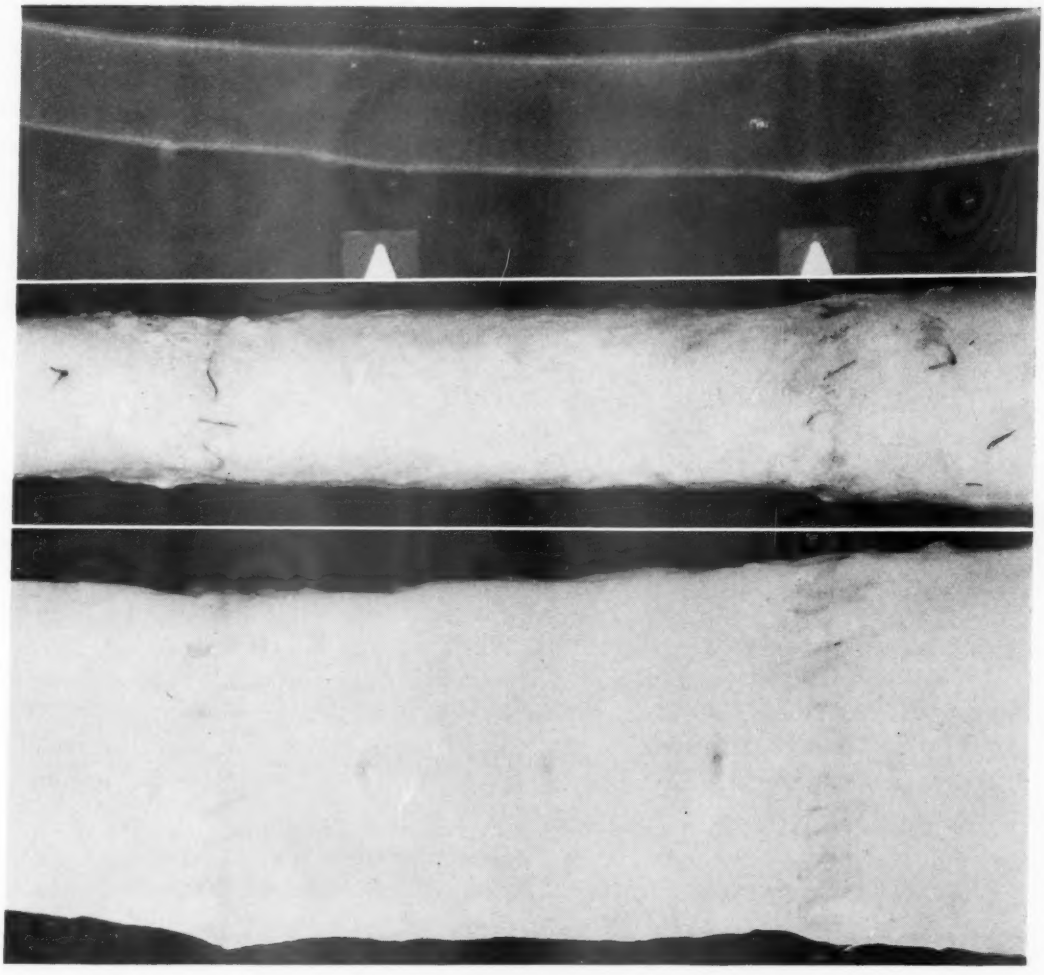

Fig. 1.—Representative fresh aortic autografts in thoracic aorta of growing pig. Upper and middle panels, respectively, show roentgenograph and photograph of a graft, 7.30 cm. long, inflated with air to 120 mm. Hg after 253 days' implantation in aorta. Note absence of both calcification and dimensional disproportion in this graft. Lower panel shows photograph of open view of a different graft, 8.70 cm. long, after 200 days' implantation in aorta.

were slaughtered. At the time of slaughter most pigs had grown from their operative weight of about 35 pounds to approximately 300 pounds. At time of death the thoracic aorta was removed, cleaned, inflated to 120 mm. Hg with air, and measured. Then this specimen in sequence was roentgenographed, photographed, inspected, and sectioned for microscopic study by either the dry celloidin or the paraffin technique.

Calcification of the graft, based upon the percentage area of the wall involved, was graded as slight if under 5 per cent, as moderate if between 5 and 20 per cent, and as severe if over 20 per cent.

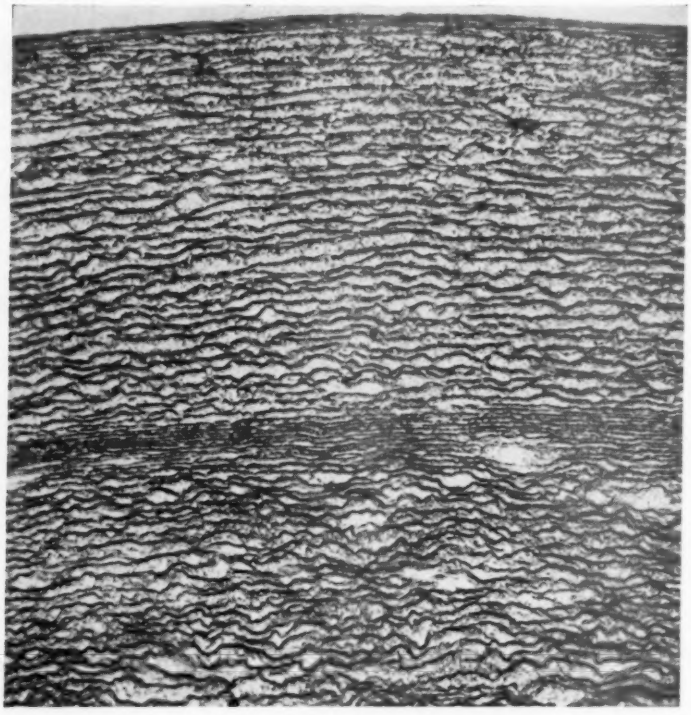

Fig. 2.—Photomicrograph of Weigert's elastic tissue stain of a longitudinal celloidin section at ×130 of a representative fresh aortic autograft implanted in the thoracic aorta of a growing pig for 195 days, showing the fiber framework of the normal aorta to have been maintained. Hematoxylin and eosin stains revealed the cellular viability of the graft to have been maintained as well.

The vascular grafts in the dogs were implanted and studied in a similar manner to that described for the thoracic aorta grafts of the pigs.

The 230 grafts in this study fall into fifteen different categories, which will now be considered individually.

TABLE I. FRESH AORTIC AUTOGRAFTS IN THORACIC AORTA OF GROWING PIG

	TOTAL GRAFTS	COMPLICATIONS	TRUE GROWTH	LOSS OF CELLULAR VIABILITY	LOSS OF FIBER CONTINUITY
Number	5	0	5	0	0
Per cent of total	100	0	100	0	0

1. *Fresh Aortic Autografts.*—A fresh thoracic aorta autograft was fashioned in each of 5 young pigs which subsequently came to slaughter by freeing, biterminally dividing, and then re-establishing aortic continuity by anastomosing the divided ends. Data of these grafts are in Table I. The grafts were implanted for an average period of 201 days, during which time the increase in weight (33 to 307 pounds), aortic diameter, *graft diameter,* aortic length, *graft length,* and crown-rump length of the pigs averaged 830,

82, *81,* 77, *76,* and 83 per cent, respectively. The gross and microscopic features of these grafts, shown in Figs. 1 and 2, reveal them to resemble the host aorta in all obvious respects. In brief, they were perfect grafts.

2. *Fresh Aortic Homografts.*—Standard (6 cm. length) thoracic aorta homograft segments were removed, placed in saline solution, and within three

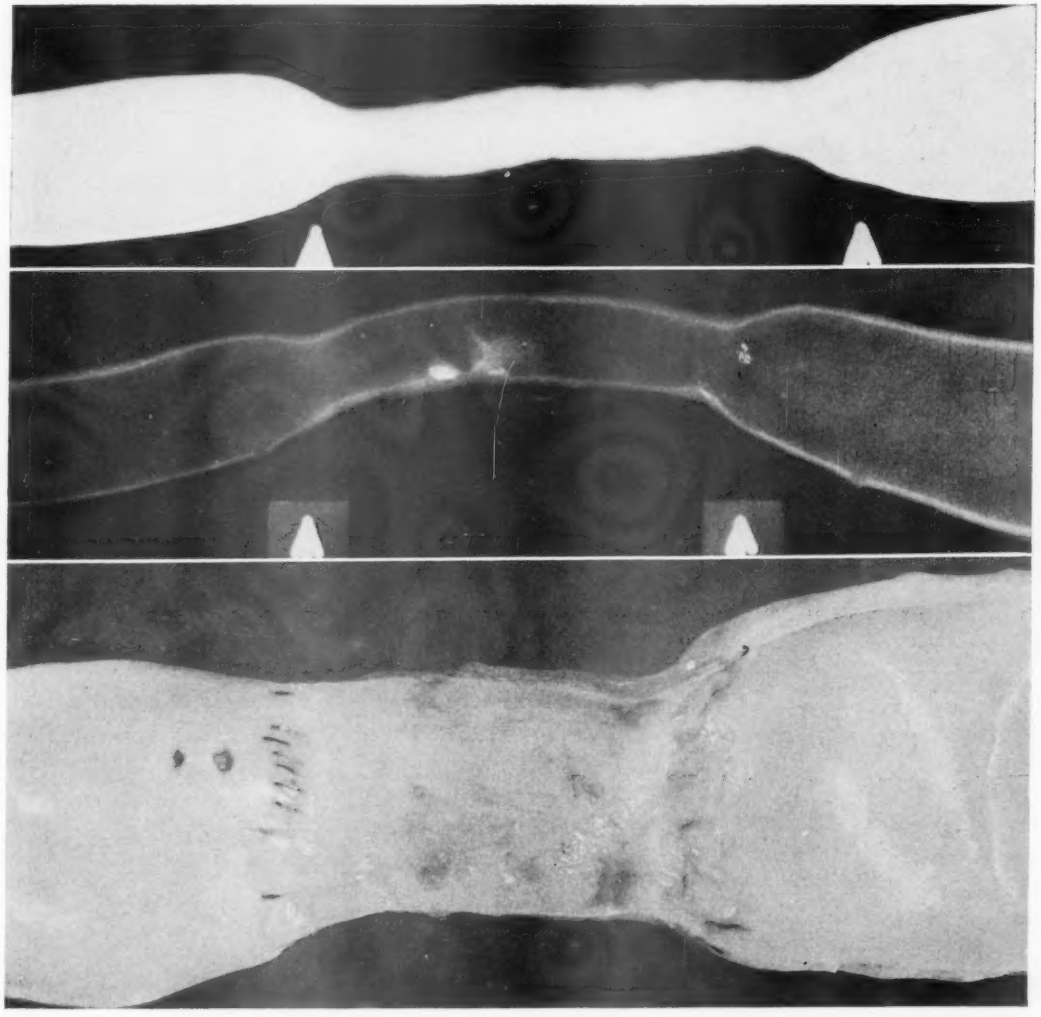

Fig. 3.—Representative fresh aortic homograft in thoracic aorta of growing pig. Upper panel shows roentgenograph of a graft, 7.0 cm. long, inflated with diodrast after 244 days in aorta. Note the dimensional disproportion between graft and aorta. The middle panel shows roentgenograph of a graft, 7.7 cm. long, inflated with air to 120 mm. Hg after 288 days in aorta. Note the slight degree of calcification present. The lower panel shows photograph of open view of a graft, 6.5 cm. long, after 211 days in aorta.

hours used to bridge aortic defects. Of 16 young pigs receiving such grafts in the thoracic aorta, 13 came to slaughter, while 3 died before that time, 2 of pneumonia and 1 from cardiac failure.

Only the grafts of the 13 pigs coming to slaughter will be considered further. Data of these grafts are in Table II. The grafts were implanted for an average period of 223 days, during which time the increase in weight

(39 to 309 pounds), aortic diameter, *graft diameter,* aortic length, *graft length,* and crown-rump length of the pigs averaged 692, 96, *15,* 93, *24,* and 69 per cent, respectively. The gross features of representative grafts are shown in Fig. 3.

The dimensional increase of these grafts was similar to that of fresh thoracic aorta segments inflated from 120 to 600 mm. Hg (Fig. 4). A spatial

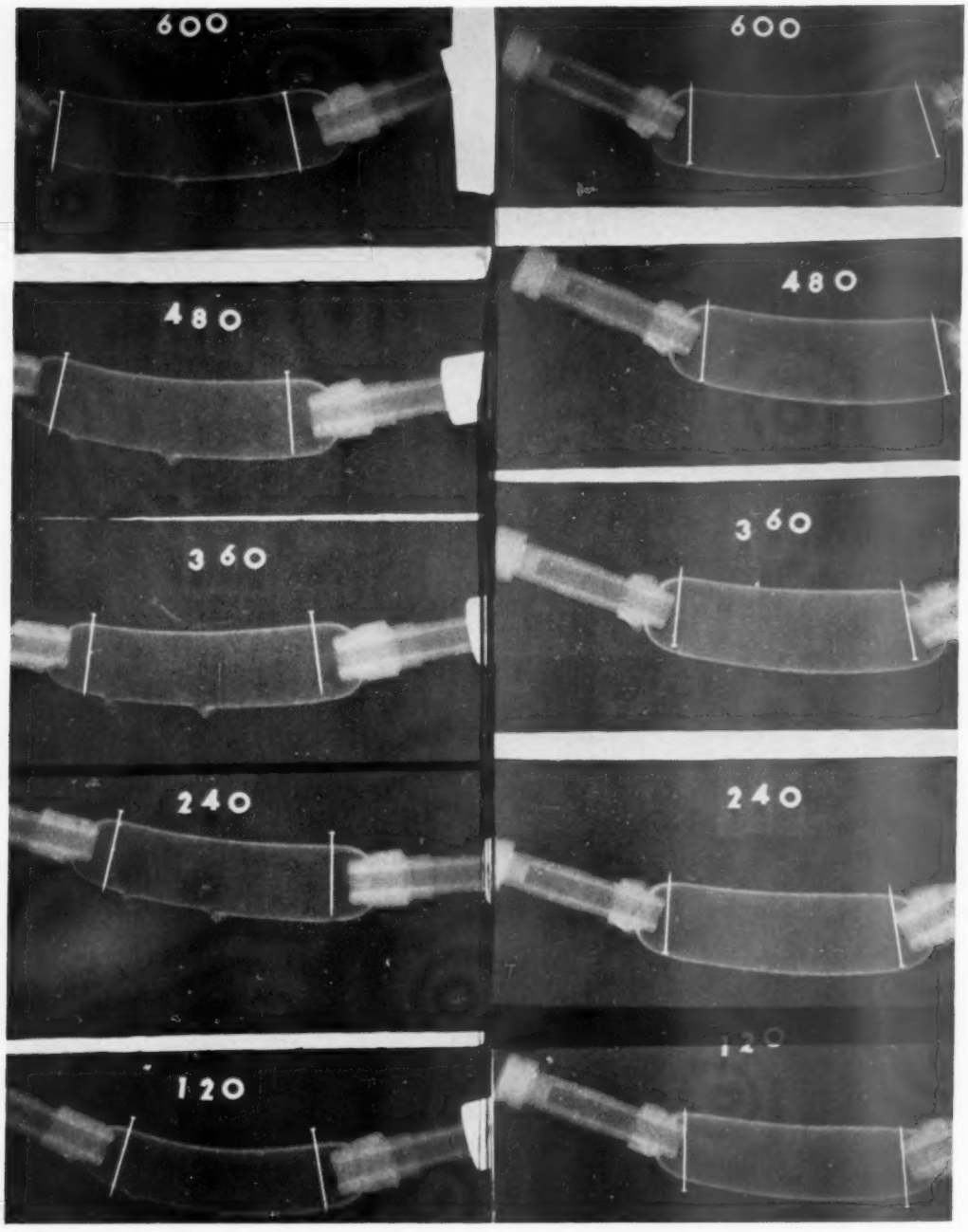

Fig. 4.—This figure of serial roentgenographs shows the acute effect upon dimensions of inflating two fresh porcine thoracic aorta segments with air to pressures as indicated. The dimensions of these segments at 600 mm. Hg were approximately 20 per cent greater than at 120 mm. Hg.

TABLE II. FRESH AORTIC HOMOGRAFTS IN THORACIC AORTA OF GROWING PIG COMING TO SLAUGHTER

	TOTAL GRAFTS	OCCLUSIVE THROMBOSIS		TRUE GROWTH	CONTRACTURE	RUPTURE OR ANEURYSM FORMATION	SEVERE CALCIFICATION	LOSS OF CELLULAR VIABILITY	LOSS OF FIBER CONTINUITY
		EARLY	LATE						
Number	13	0	0	0	0	0	1	13	0
Per cent of total	100	0	0	0	0	0	8	100	0

rearrangement of the arterial fiber framework could account for dimensional changes of this magnitude (20 per cent). Fresh aortic homografts appear incapable of true growth. This seems at variance with the findings of Sauvage and Harkins (1953) that 1 to 3 cm. length aortic homografts in the abdominal aorta of growing pigs increased in diameter but at a slower rate than the host

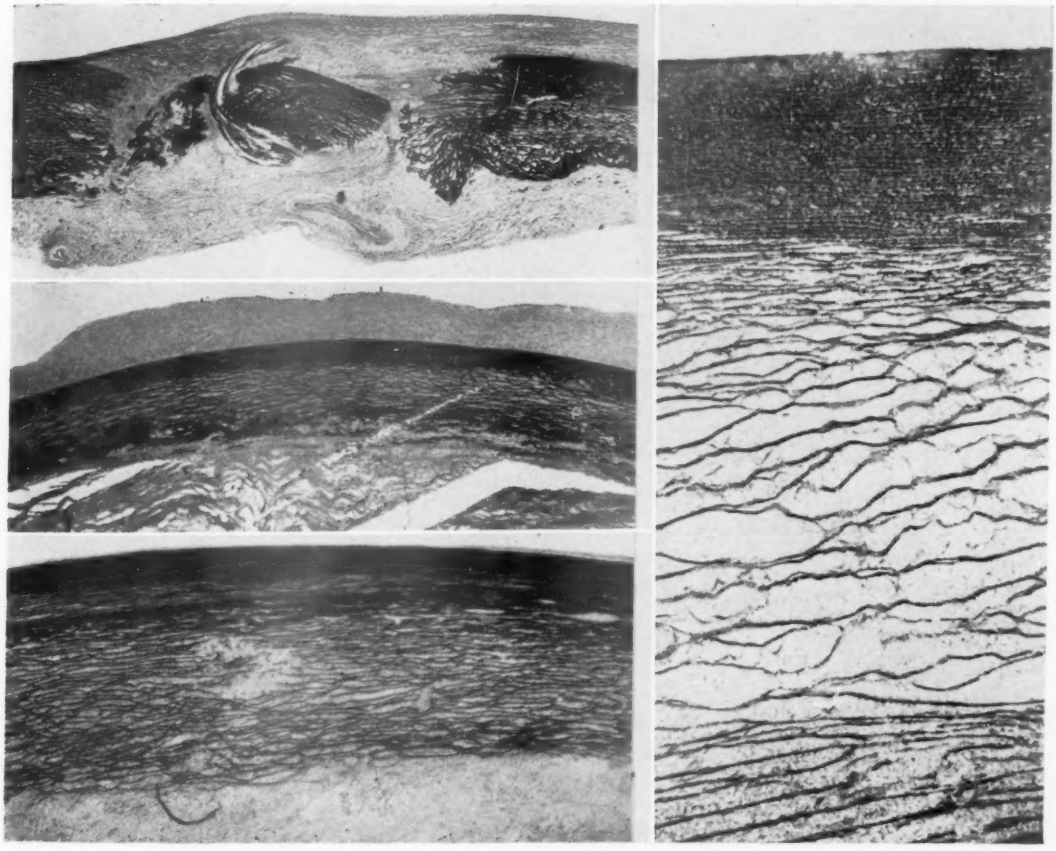

Fig. 5.—The healing of aortic homografts as illustrated by photomicrographs of Weigert's elastic tissue stains of longitudinal celloidin sections of representative fresh aortic homografts implanted in the thoracic aorta of growing pigs for an average of 234 days. Upper panel on left of lower anastomosis at ×30 reveals host-graft junction to have been healed by ingrowth of fibrous elements of the aortic and periaortic tissues with extension of this tissue along the surface of the fiber framework of the graft as an inner layer. Middle panel on left of wall 1 cm. from upper anastomosis at ×30 shows the presence of a thick inner layer atop the condensed inner portion of the fiber framework of the original graft. Lower panel on left of wall 2 cm. from upper anastomosis at ×45 shows the inner layer to be practically absent at this point. Single panel on right of central portion of graft at ×130 shows the surface of the condensed, acellular inner portion of the fiber framework of the original graft to be without covering.

aorta. However, these findings are in general agreement. The present study of longer-length, larger-caliber grafts merely enables the actual results to be seen more clearly.

The microscopic features of representative grafts are shown in Fig. 5. The original graft, although losing cellular viability, maintained gross fiber continuity and came to be incorporated by autogenous fibrous tissue. The healed graft thus consisted of an autogenous fibrous tissue tube with a foreign (homologous) protein fiber framework. The inner part of this framework was condensed and acellular, while its outer part was infiltrated by fibrous elements. The residual fiber framework of the original graft was covered internally by a fibrous tissue layer which thinned toward the middle and often was absent centrally. The inner and outer layers of the healed graft did not take the elastic tissue stain. However, the superficial portion of the fibrous tissue uniting the host and graft took the elastic tissue stain and appeared to originate from the aortic intima. The outer layer of the graft appeared to originate from the periarterial tissues, while its inner layer appeared to originate from both the fibrous tissue at the anastomotic site and the extensions of the outer coat growing in through the interstices of, or minute defects in, the fiber framework of the original graft. Calcification of the condensed, acellular portion of the fiber framework of the original graft occurred in 11 of 13 grafts and was graded as slight in 8, as moderate in 2, and as severe in 1 instance.

The persistence of the fiber framework of the original graft in the absence of cellular viability prevented significant dimensional changes of either a pathologic or a physiologic nature. Two grafts had unusual thinning of their fiber frameworks, and both showed moderate dilatation.

It is of considerable interest that the condensed, acellular mass of homologous elastic tissue which formed the inner portion of the homograft wall still possessed significant elasticity at time of slaughter. Removal of adherent host fibrous tissue was usually necessary in order to allow demonstration of this property.

In summary, these grafts, although losing cellular viability, provided stable fiber frameworks for ingrowth of autogenous fibrous elements. They were neither subject to aneurysm formation nor able to respond to growth stimulation. Their incidence of calcification was high, but its degree was seldom severe. (The collective fate of these fresh homografts is superior to that of those frozen, dried, or frozen-dried homografts which are described, respectively, in sections 3, 4, and 5.)

TABLE III. FROZEN AORTIC HOMOGRAFTS IN THORACIC AORTA OF GROWING PIG

	TOTAL GRAFTS	OCCLUSIVE THROMBOSIS		TRUE GROWTH	CONTRACTURE	ANEURYSM FORMATION	SEVERE CALCIFICATION	LOSS OF CELLULAR VIABILITY	LOSS OF FIBER CONTINUITY
		EARLY	LATE						
Number	12	0	1	1	0	1	4	11	1
Per cent of total	100	0	8	8	0	8	33	92	8

3. *Frozen Aortic Homografts.*—Standard fresh thoracic aorta homograft segments were placed in Pyrex tubes, frozen by immersion in dry ice–alcohol baths for one hour, and then rapidly thawed and used to bridge aortic defects. Of 12 young pigs receiving such grafts in their thoracic aorta, 11 came to

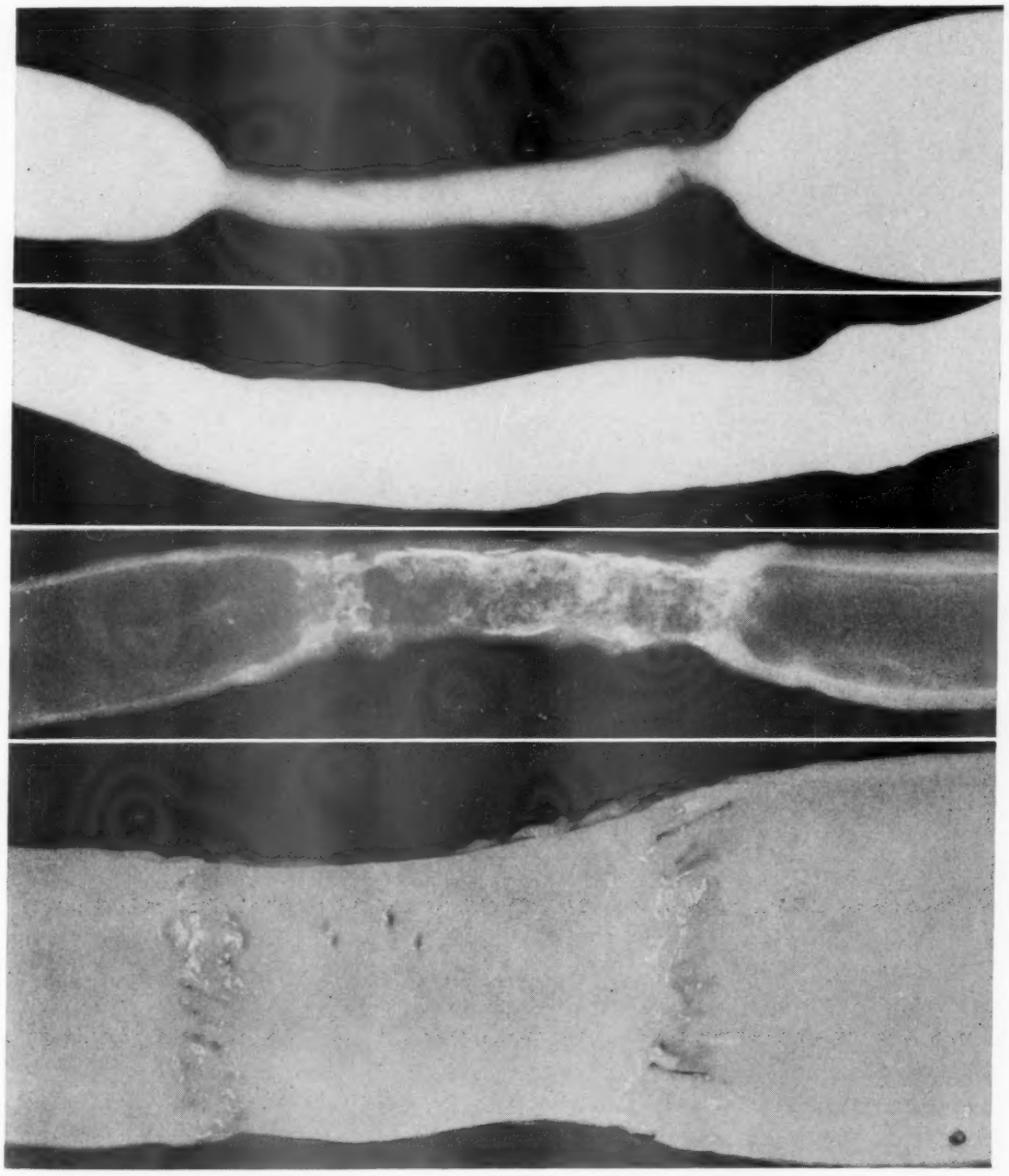

Fig. 6.—Frozen aortic homografts in thoracic aorta of growing pig. Upper panel shows roentgenograph of a graft, 7.30 cm. long, inflated with Diodrast after 232 days in aorta. Note the dimensional disproportion between graft and aorta. The microscopic picture of this graft is shown in the upper panel of Fig. 7. Second panel shows roentgenograph of a graft, 11 cm. long, inflated with Diodrast after 173 days in aorta. Such enlargement of the graft is the exception rather than the rule. Note the microscopic picture of this graft in the lower panel of Fig. 7. Third panel shows roentgenograph of a graft, 6.20 cm. long, after 189 days in the aorta. This degree of calcification, classified as severe, developed in 4 of the 12 grafts in this series. Lower panel shows photograph of an opened graft, 7.80 cm. long, after 193 days in aorta. This exceptional graft survived both freezing and homotransplantation to remain a living artery.

slaughter. Data of these grafts are in Table III. The grafts were implanted for an average period of 194 days, during which time the increase in weight (36 to 302 pounds), aortic diameter, *graft diameter,* aortic length, *graft length,* and crown-rump length of the pigs averaged 739, 106, *17,* 88, *32,* and 74 per cent, respectively. The gross features of four grafts are shown in Fig. 6. Note the marked difference in size of the two grafts inflated with Diodrast. The smaller has not increased in size, while the larger has. Fig. 7 reveals that the fiber framework of the smaller graft is intact, while that of the larger graft is undergoing loss of continuity. The fragmentation of the fiber framework of the latter graft is assumed to have allowed its wall to respond to the forces

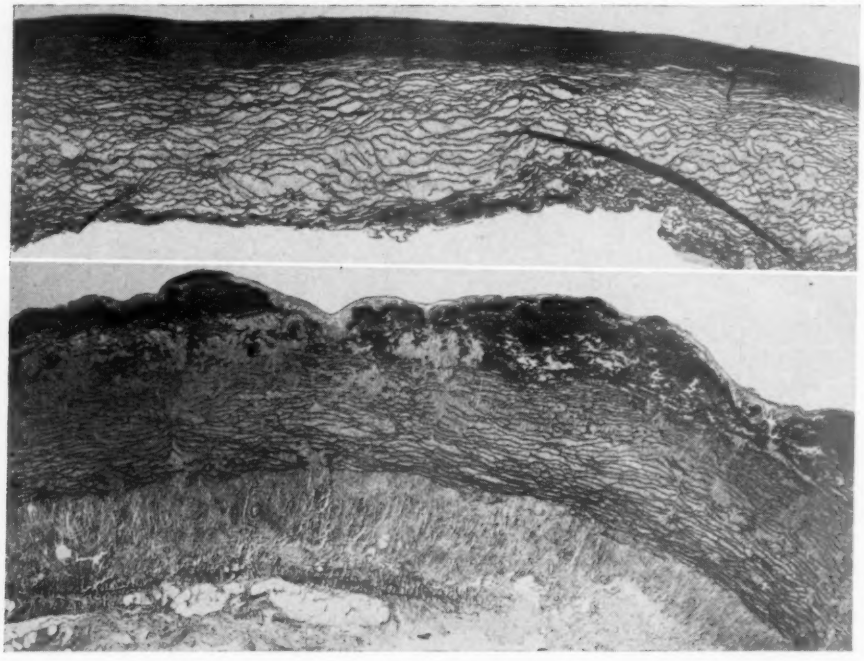

Fig. 7.—Photomicrographs of Weigert's elastic tissue stains of longitudinal celloidin sections at ×30 of the grafts shown, respectively, in the upper two panels of Fig. 6. Note that restriction of dimensional change is associated with intactness of the condensed, acellular inner portion of the fiber framework, while dimensional increase is correlated with fragmentation of this portion of the fiber framework.

acting upon it. Whether the resultant dimensional changes represent a passive response to intraluminal pressure or an active response to growth stimuli is not known. Calcification occurred in 9 of the 12 grafts and was graded as slight in 3, as moderate in 2, and as severe in 4 instances. The 33 per cent incidence of severe calcification in these grafts contrasts with an 8 per cent incidence in the fresh grafts and indicates that freezing increases the liability of fresh thoracic aorta homografts to this complication. The statistical significance of the occurrence of 1 aneurysm in this series of 12 grafts is doubtful. The same applies to the 1 graft that maintained its histologic identity as an artery. This latter graft clearly indicates that freezing per se did not destroy either the cellular viability or the fiber continuity of the grafts.

4. *Dried Aortic Homografts.*—Standard fresh thoracic aorta homograft segments were placed in Pyrex specimen tubes and dried without preliminary

freezing by attachment to a diffusion pump–doublet–liquid nitrogen vacuum system similar to that described by Hufnagel, Rabil, and Reed (1954). As a consequence of rapid evaporation, scattered areas of the vessels froze and subsequently dried by sublimation. The vessels were hermetically sealed off from the vacuum system after being run for an average period of ten hours,

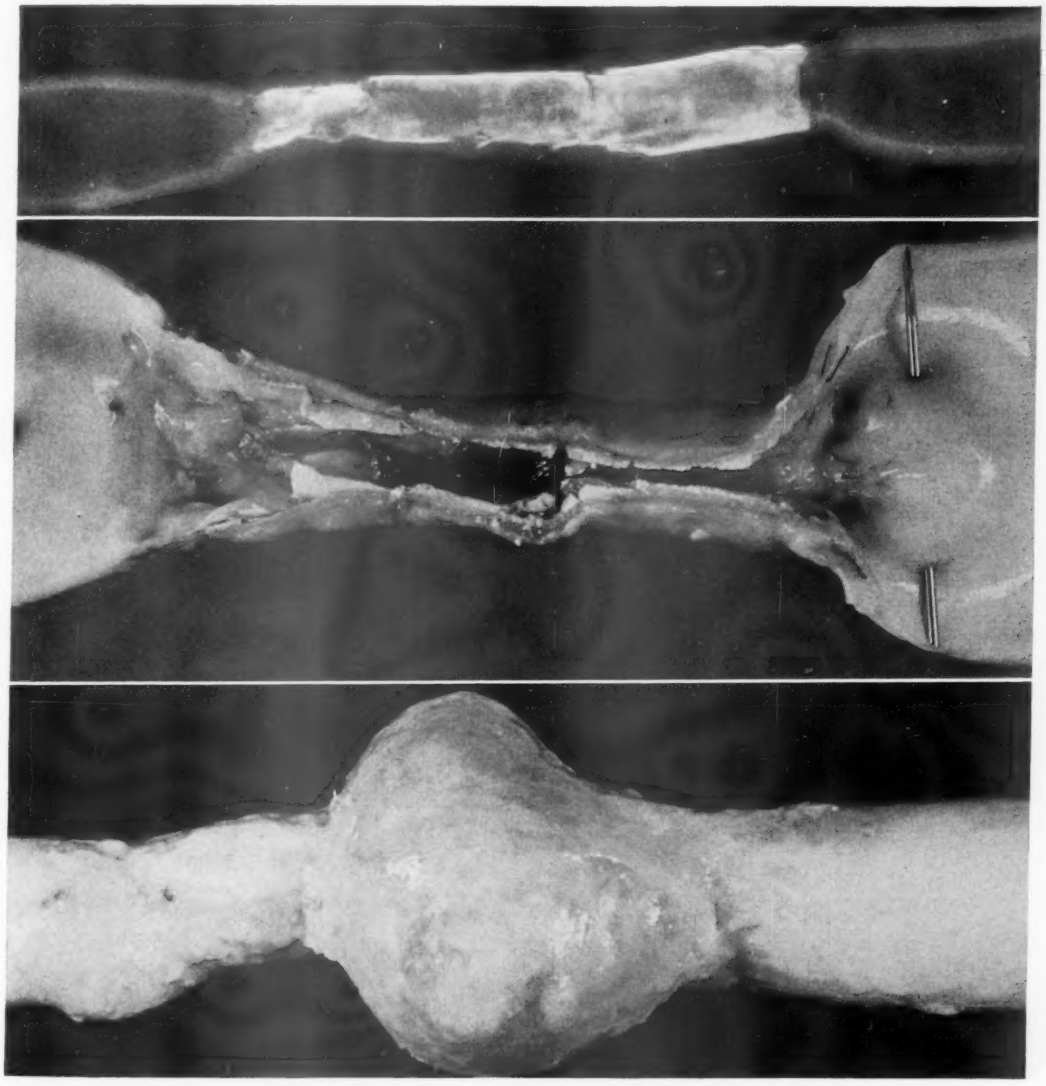

Fig. 8.—Dried aortic homografts in thoracic aorta of growing pig. Upper panel shows roentgenograph of a severely calcified graft, 8.00 cm. long, after 186 days in aorta. Such degenerative changes occurred in all 10 grafts of this series. Middle panel shows photograph of the open view of this rigid tube. Lower panel shows photograph of the aneurysmal graft in this series, 8.00 cm. long, inflated with air to 120 mm. Hg after 270 days in aorta.

during the last three of which the vessels appeared completely dry. The processed segments were wizened, hard, thin-walled, and semitransparent. After an average storage period of nine days, they were hydrated in saline solution and used to bridge aortic defects. About forty-five minutes of hydration

was required to render the vessels suitable for use as grafts. When hydrated, these segments were less elastic and tore easier than the normal aorta. However, these deficiencies led to no difficulty in using them as grafts. Of 10 young pigs receiving such grafts in their thoracic aorta, 9 came to slaughter and 1 died before that time. Data of these grafts are in Table IV. Note that all grafts developed severe calcific changes and that 1 lost its fiber continuity and became aneurysmal. These gross features are seen in Fig. 8. The microscopic features of these grafts, apart from the heavier calcification of the acellular, condensed inner portion of the fiber framework of the original graft, were similar to those of the fresh homografts (Fig. 5). The increase in weight (36 to 332 pounds), aortic diameter, *graft diameter,* aortic length, *graft length,* and crown-rump length of the eight pigs coming to slaughter without aneurysms averaged 820, 110, *26,* 98, *15,* and 80 per cent, respectively.

TABLE IV. DRIED AORTIC HOMOGRAFTS IN THORACIC AORTA OF GROWING PIG

	TOTAL GRAFTS	OCCLUSIVE THROMBOSIS		TRUE GROWTH	CONTRACTURE	ANEURYSM FORMATION	SEVERE CALCIFICATION	LOSS OF FIBER CONTINUITY
		EARLY	LATE					
Number	10	0	0	0	0	1	10	1
Per cent of total	100	0	0	0	0	10	100	10

The fact that drying by evaporation denatures protein is well known. Accordingly, we expected that these grafts would show severe degenerative changes. However, its extent and degree were surprising.

5. *Frozen-dried Aortic Homografts.*—Standard fresh thoracic aorta homograft segments were placed in Pyrex specimen tubes, frozen by immersion in dry ice–alcohol baths, and then dried from the frozen state by attachment to the previously mentioned diffusion pump–doublet–liquid nitrogen vacuum system. The vessels were hermetically sealed off from the system after being run for an average period of ten hours, during the last three of which the vessels appeared completely dry. The processed segments were white, brittle, and of approximately the same wall thickness as the fresh vessel. In

TABLE V. FROZEN-DRIED AORTIC HOMOGRAFTS IN THORACIC AORTA OF GROWING PIG

	TOTAL GRAFTS	OCCLUSIVE THROMBOSIS		TRUE GROWTH	CONTRACTURE	ANEURYSM FORMATION	SEVERE CALCIFICATION	LOSS OF FIBER CONTINUITY
		EARLY	LATE					
Number	16	0	0	0	0	0	5	0
Per cent of total	100	0	0	0	0	0	31	0

order to determine accurately the drying conditions obtained under these circumstances, runs were performed with similar specimens in tubes having sidearms at specimen level, to which continuously recording Pirani gauges were attached. The absolute pressure at this level did not exceed 150 microns of mercury and at the termination of the run had been stable at or near 1 micron for three to four hours. The final moisture content of several specimens was determined by the ninety-six-hour phosphorus pentoxide vacuum desiccator technique and found to be at or within a few hundredths of 1 per cent.

After an average storage period of seventy-five days, these frozen-dried aortic homografts were hydrated in saline solution and used to bridge aortic defects. About twenty-five minutes of hydration was required to render the vessels suitable for use as grafts. When hydrated, these segments had physical properties characteristic of the fresh state. Of 16 young pigs receiving such grafts in their thoracic aorta, 11 came to slaughter and 5 died before that time.

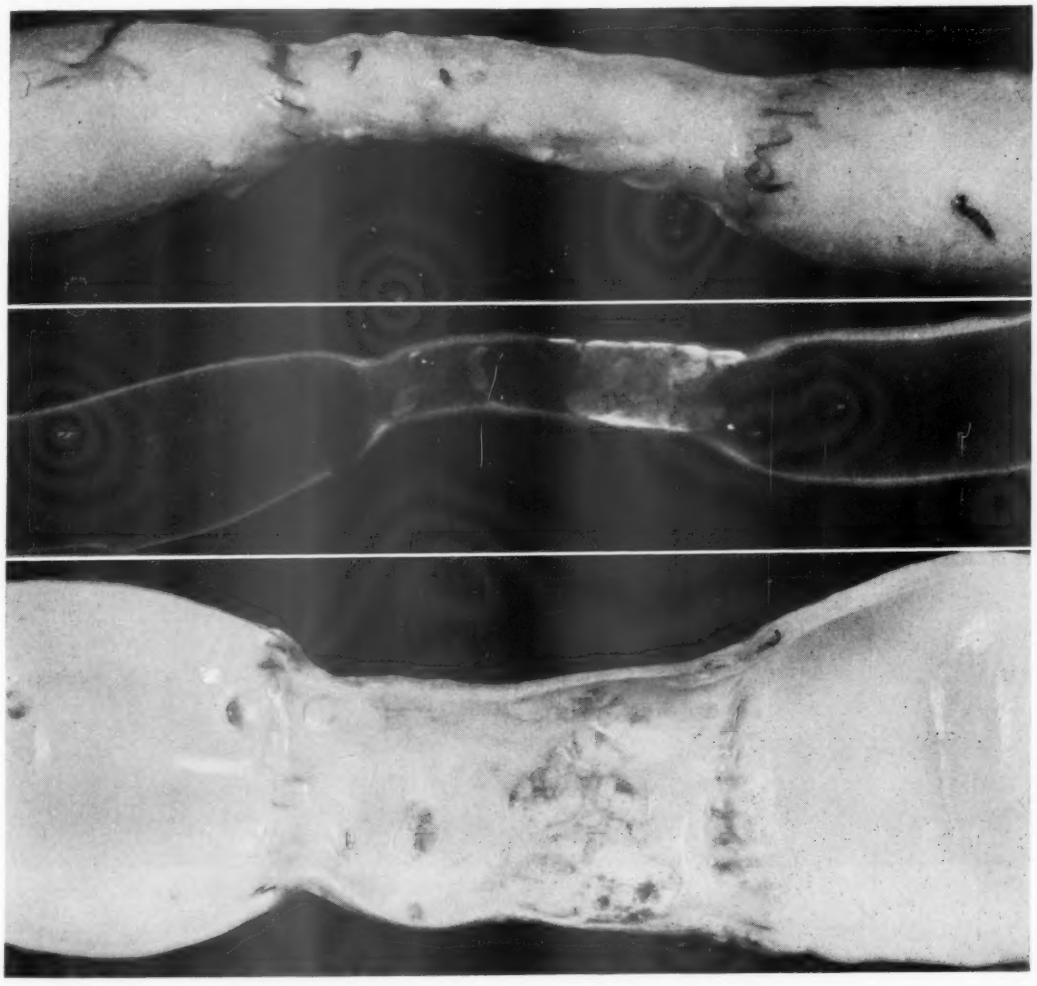

Fig. 9.—A 152-day-old frozen-dried aortic homograft, 6.5 cm. long, in thoracic aorta of growing pig for 191 days. Upper and middle panels, respectively, show photograph and roentgenograph of this graft inflated with air to 120 mm. Hg. The calcification visible in the middle panel is classified as of moderate degree. Lower panel shows photograph of open view of this same graft.

Data of these grafts are in Table V. Calcification occurred in 15 of 16 grafts and was graded as slight in 3, as moderate in 7, and as severe in 5 instances. The 5 pigs dying before slaughter survived an average of ninety-nine days after operation, while the remaining 11 pigs came to slaughter an average of 218 days after operation. These latter pigs increased in weight (40 to 290 pounds), aortic diameter, *graft diameter,* aortic length, *graft length,* and crown-

rump length an average of 625, 100, *24*, 100, *27*, and 69 per cent, respectively. The gross features of a representative graft are shown in Fig. 9. The incidence (31 per cent) of severe calcification developing in these grafts is similar to that (33 per cent) of the frozen grafts. This similarity indicates that the additional procedure of drying by sublimation did not increase the liability of frozen grafts to severe calcific change. Freezing by our method definitely increased the liability of fresh homografts to severe calcification (8 per cent as compared to 33 per cent). However, the deleterious effects of drying without prior freezing are amply demonstrated by the 100 per cent incidence of severe calcific change occurring in the dried homograft group.

Fig. 10.—Representative Tyrode's and frozen-dried aortic homograft composite grafts in thoracic aorta of growing pig. Upper panel shows photograph of a composite graft inflated with air to 120 mm. Hg after 231 days in aorta. Note the dimensional disproportion between the aorta and the graft. Lower panel shows roentgenograph of composite graft after 215 days in aorta. Note the severe calcification in the lower (frozen-dried) portion of this graft.

The histologic fate of these frozen-dried homografts, apart from their greater degree of calcific change, was very similar to that of the fresh homografts (Fig. 5). Neither frozen nor frozen-dried aortic homografts were as satisfactory as fresh aortic homografts in our studies. However, banks are essential if homografts are to be available clinically. The frozen-dried artery bank represents an answer to a pressing storage problem and, as such, it constitutes a significant advance in the field of reconstructive arterial surgery. Sauvage, Wesolowski, and Pinc (1955) have demonstrated that satisfactory frozen-dried arteries may be prepared by simple techniques with apparatus that is easily available to any community having need for an artery bank.

6. *Tyrode's and Frozen-dried Aortic Homograft Composite Grafts.*—Standard fresh thoracic aorta homograft segments were divided into halves, which were preserved for periods up to five days and then anastomosed to re-form the segments. These were then used to bridge aortic defects. The halves were preserved either by maintenance at 4° C. in Tyrode's solution containing 10 per cent homologous serum or by freeze-drying as previously described. Different halves of successive homograft segments were alternately preserved by these methods. Of 14 young pigs receiving such composite grafts in their thoracic aorta, 10 came to slaughter and 4 died within thirty days. Only the grafts of the pigs coming to slaughter will be considered further. These were implanted for an average period of 228 days, during which time the pigs increased in weight an average of 868 per cent (31 to 300 pounds). The dimensional changes of this composite homograft group (Fig. 10) were similar to those of the other groups previously detailed. Data of these composite grafts are in Table VI. Note that in no instance did the frozen-dried segment of the composite graft show less calcification than the Tyrode's segment, while in 70 per cent of instances the frozen-dried segment showed more calcification. This latter circumstance is shown in Fig. 10. These data clearly indicate that our frozen-dried grafts had more calcification than did grafts preserved for a few days in Tyrode's solution. The data of the previous sections implicate freezing rather than sublimation as the factor responsible for this increased liability of frozen-dried grafts to calcific change.

TABLE VI. TYRODE'S AND FROZEN-DRIED HOMOGRAFT COMPOSITE GRAFTS IN THORACIC AORTA OF GROWING PIG COMING TO SLAUGHTER

	TOTAL GRAFTS	CLOSED	OPEN	CALCIFICATION IN FROZEN-DRIED SEGMENT COMPARED TO TYRODE'S SEGMENT		
				LESS	SAME	MORE
Number	10	3	7	0	3	7
Per cent of total	100	30	70	0	30	70

On the basis of analytic data to be reported by Wesolowski, Sauvage, and Pine (1955) concerning the drying characteristics of several vacuum systems, including the one employed for these studies, we believe that the frozen grafts of this group were dried in an optimal manner. Our data regarding calcification speak against the opinion that frozen-dried arterial homografts are better than fresh arterial homografts; and, until frozen-dried grafts are shown to be superior to fresh grafts by carefully controlled comparisons using either our method or the alternate experiment method, we shall continue to regard these data as valid. We believe the fresh graft to be best and that study directed toward permanently maintaining the viability of homografts living at time of implantation will prove of more ultimate significance than will those directed toward decreasing the antigenicity of homografts nonviable at implantation.

Sauvage and Harkins (1953) found the incidence of calcification of preserved (Tyrode's solution) short-length aortic homografts in the abdominal aorta of the growing pig to be low and uninfluenced by periods of preservation

from one to 120 days. Older grafts were not studied. Our data do not allow comment as to whether the same results would obtain in the thoracic aorta. The work of Kanar and associates (1954) suggests that they would not. However, on the basis of our data, we recommend that if a patient who may need an arterial graft is to be operated upon within a few days after sterile arterial segments have been procured, these segments should be preserved in Tyrode's solution until the operation and then used as grafts if needed. If there is not

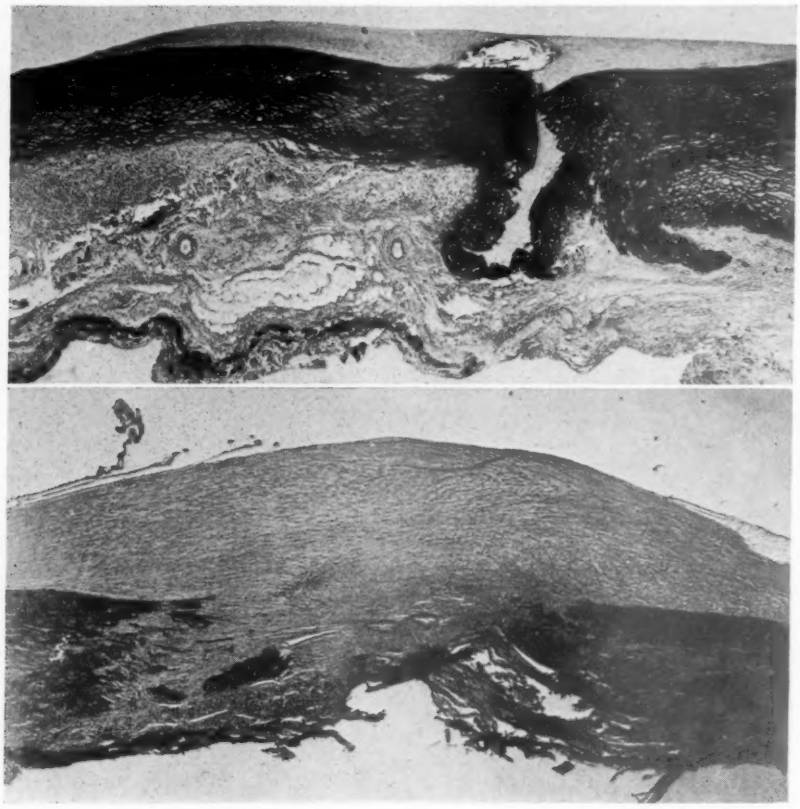

Fig. 11.—Photomicrographs of Weigert's elastic tissue stains of celloidin sections at ×30 across the intergraft anastomoses of two Tyrode's and frozen-dried aortic homograft composite grafts in thoracic aorta of growing pig. Upper panel shows intergraft anastomosis after thirty days in aorta. The frozen-dried graft is to the left. Note the ingrowth of fibrous elements through the anastomotic junction. Lower panel shows intergraft anastomosis after 246 days in aorta. The frozen-dried graft is to the right. Although the outer coat of fibrous tissue has been dissected away, the anastomotic junction can be seen to be firmly united by ingrowth of fibrous elements of the periarterial tissues.

an early need for grafts or if these segments are not used after having been kept for a few days in the fluid medium, our policy is to freeze-dry them (Sauvage, Wesolowski, and Pinc, 1955).

The microscopic features of both components of these composite grafts closely resembled those of fresh homografts (Fig. 5) except for the heavy calcification in the acellular, condensed elastic tissue remnant of the frozen-dried graft. The intergraft anastomoses were distinguished from the adjacent portion of each graft by the presence of an inner layer which was continuous with the periarterial tissues through the medium of the fibrous tissue stock uniting

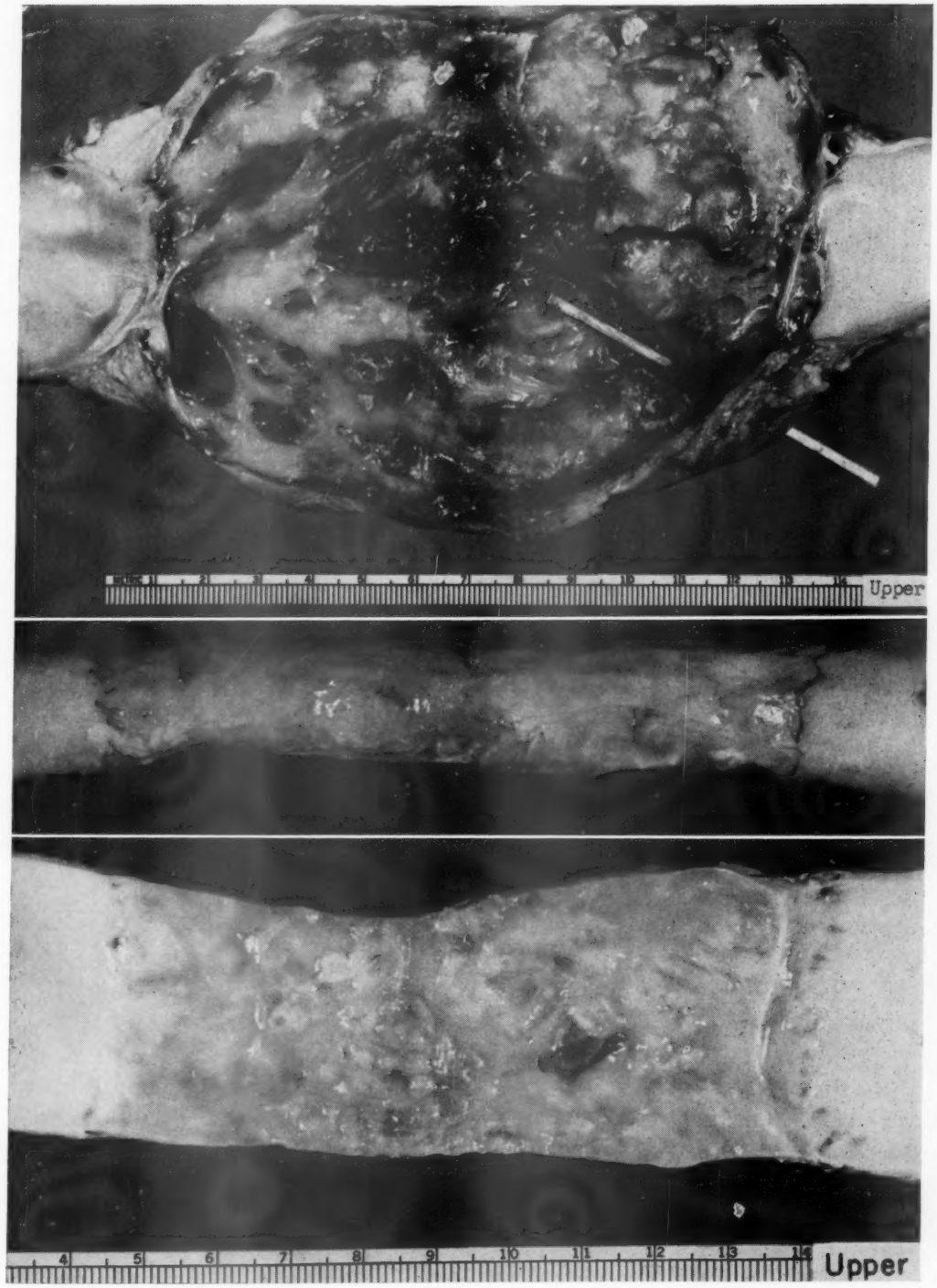

Fig. 12.—Representative fresh canine aortic heterografts in thoracic aorta of growing pig. Upper panel shows photograph of open view of an aneurysm which ruptured 134 days after graft implantation. Middle and lower panels show photographs of inflated (with air to 120 mm. Hg) and open views of a graft after 287 days in aorta. The microscopic picture of this latter graft is shown in Fig. 13.

the graft ends. The inner layer extended from the anastomotic site along the surface of the grafts but thinned rapidly to disappear within a few millimeters. These microscopic features are shown in Fig. 11. The fibrous tissue at the intergraft anastomosis differed from that at the host-graft anastomoses by the absence of elements taking the elastic tissue stain. The elastic tissue remnant of the original graft had an inner layer at and adjacent to any area where the fibrous elements of the host could make their way to the surface. This occurred at the host-graft and intergraft anastomoses and at those sites along the graft where its original fiber framework remained pervious to the ingrowth of fibrous elements or became so subsequently.

TABLE VII. FRESH CANINE AORTIC HETEROGRAFTS IN THORACIC AORTA OF GROWING PIG

	TOTAL GRAFTS	PIGS DYING OF RUPTURED ANEURYSM	PIGS COMING TO SLAUGHTER			LOSS OF CELLULAR VIABILITY	LOSS OF FIBER CONTINUITY
			WITH ANEURYSM	WITHOUT ANEURYSM			
				OPEN	THROMBOSED		
Number	10	5	1	4	0	10	10
Per cent of total	100	50	10	40	0	100	100

7. *Fresh Canine Aortic Heterografts.*—These segments were prepared in the same manner as the fresh aortic homografts. Of 10 young pigs receiving such grafts in their thoracic aorta, 5 died of ruptured aneurysms and 5 came to slaughter after an average period of eighty-four and 266 days, respectively. Data of these grafts are in Table VII. Note that while 6 of 10 grafts formed aneurysms (5 of which ruptured!), only 1 of the 5 coming to slaughter formed an aneurysm. The gross features of representative grafts are shown in Fig. 12.

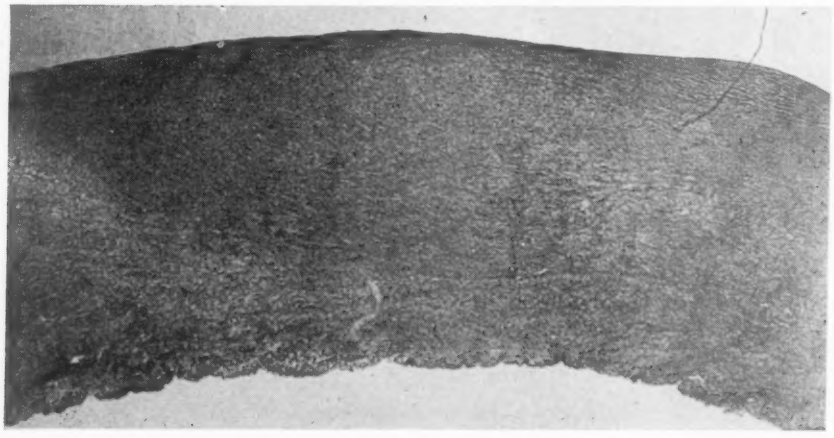

Fig. 13.—Photomicrographs of Weigert's elastic tissue stain of longitudinal celloidin section at ×30 of the fresh canine aortic heterograft shown in the lower two panels of Fig. 12. Note the complete absence of elastic tissue. Apparently, the autogenous fibrous tissue ingrowth had attained sufficient strength to resist adequately the intraluminal pressure by the time the continuity of the fiber framework of the original graft was lost.

The 5 pigs coming to slaughter increased in weight (28 to 306 pounds), aortic diameter, *graft diameter,* aortic length, *graft length,* and crown-rump length an average of 993, 107, *170,* 95, *60,* and 99 per cent, respectively. The dimensional increase of the four grafts coming to slaughter without aneurysmal change was similar to that of the aorta.

The microscopic features of a representative graft are shown in Fig. 13. The original graft lost both its cellular viability and gross fiber continuity. Indeed, only calcified fragments of elastic tissue were found in 4 of the 5 grafts coming to slaughter. The original graft had, in effect, been entirely replaced by autogenous fibrous tissue. The healing pattern of these heterografts appeared to be that of the homografts adapted to the breakdown of the fiber framework of the original graft.

In summary, the consistent breakdown of the structure of these grafts often led to aneurysm formation and rupture. However, in some instances the autogenous fibrous tissue replacement formed a stable conduit.

8. *Frozen-dried Canine Aortic Heterografts.*—These segments were prepared in the same manner as the frozen-dried homografts. Of 12 young pigs receiving such grafts in their thoracic aorta, 2 died with aneurysms at twenty-nine and sixty-two days after operation, respectively, and 10 came to slaughter after an average of 231 days, with a weight gain averaging 850 per cent (30 to 285 pounds). Data of these grafts are in Table VIII. Note that only 3 of 12 grafts became aneurysmal, as compared to 6 of 10 in the fresh canine heterograft group. Also, in contrast to the patency of all 5 of the fresh canine heterografts coming to slaughter, 4 of 9 (44 per cent) of the frozen-dried grafts were completely thrombosed at slaughter. Thus, the combined incidence of thrombosis and aneurysm formation in the frozen-dried group (7 of 12 or 58 per cent) is similar to the incidence of aneurysm formation in the fresh group (6 of 10 or 60 per cent). The gross features of 4 frozen-dried canine heterografts are shown in Fig. 14.

TABLE VIII. FROZEN-DRIED CANINE AORTIC HETEROGRAFTS IN THORACIC AORTA OF GROWING PIG

	TOTAL GRAFTS	PIGS DYING WITH ANEURYSM	PIGS COMING TO SLAUGHTER			LOSS OF FIBER CONTINUITY
			WITH ANEURYSM	WITHOUT ANEURYSM		
				OPEN	THROMBOSED	
Number	12	2	1	5	4	11
Per cent of total	100	17	8	42	33	92

Microscopically, the pattern of healing in these grafts was the same as that previously described for the other types of grafts. Although fiber continuity was lost in 11 of 12 grafts, the disappearance of elastic tissue was relatively complete in only 1 instance. This is in marked contrast to the nearly complete disappearance of elastic tissue which occurred in the fresh canine heterografts. Freeze-dry processing appeared to increase the resistance of the canine aortic heterograft to fragmentation and subsequent absorption by the host. The relative infrequency of aneurysm formation in these grafts suggests that the loss of fiber continuity of most grafts occurred after the autogenous fibrous tissue ingrowth had attained considerable strength. Fiber framework degeneration at such a time could account for the high incidence of delayed thrombosis seen in this group. The microscopic appearance of fiber frameworks in different stages of disintegration are shown in Fig. 15.

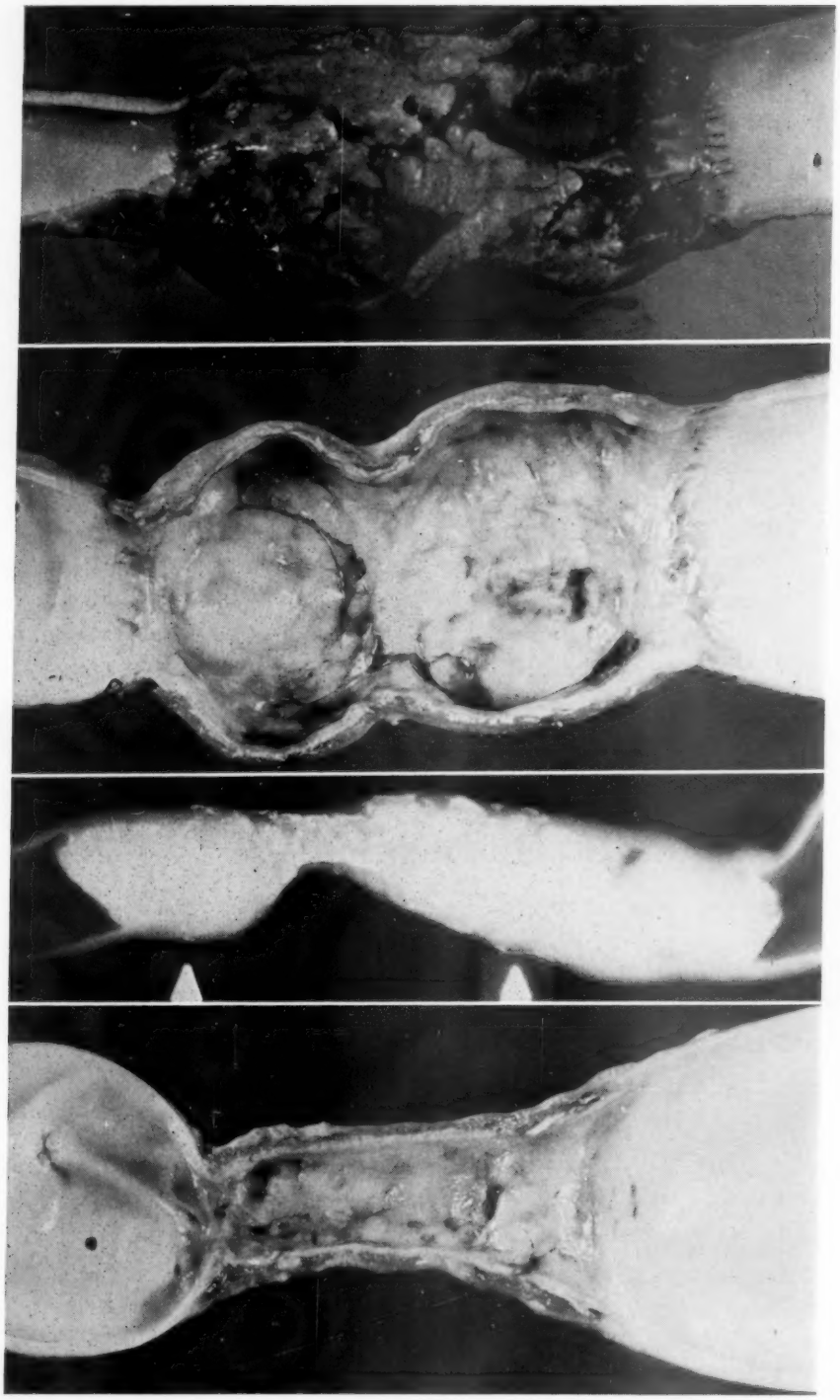

Fig. 14.—Frozen-dried aortic heterografts in thoracic aorta of growing pig showing the diverse gross morphologic fates of these grafts. Upper panel shows photograph of open view of an aneurysm, 9 cm. long, which ruptured thirty-two days after graft implantation. Second panel shows photograph of open view of a moderate-sized aneurysm, 10.0 cm. long, developing in a graft implanted in aorta for 186 days. Third panel shows roentgenograph of a graft, 6.0 cm. long, after 241 days in aorta which had undergone delayed thrombosis. Note the proximal and distal extensions of the thrombus toward the nearest collateral vessels. Lower panel shows photograph of open graft, 7.0 cm. long, after 231 days in aorta. The fiber framework of this graft has been maintained (Fig. 15), while those of the preceding 3 grafts in this illustration have not.

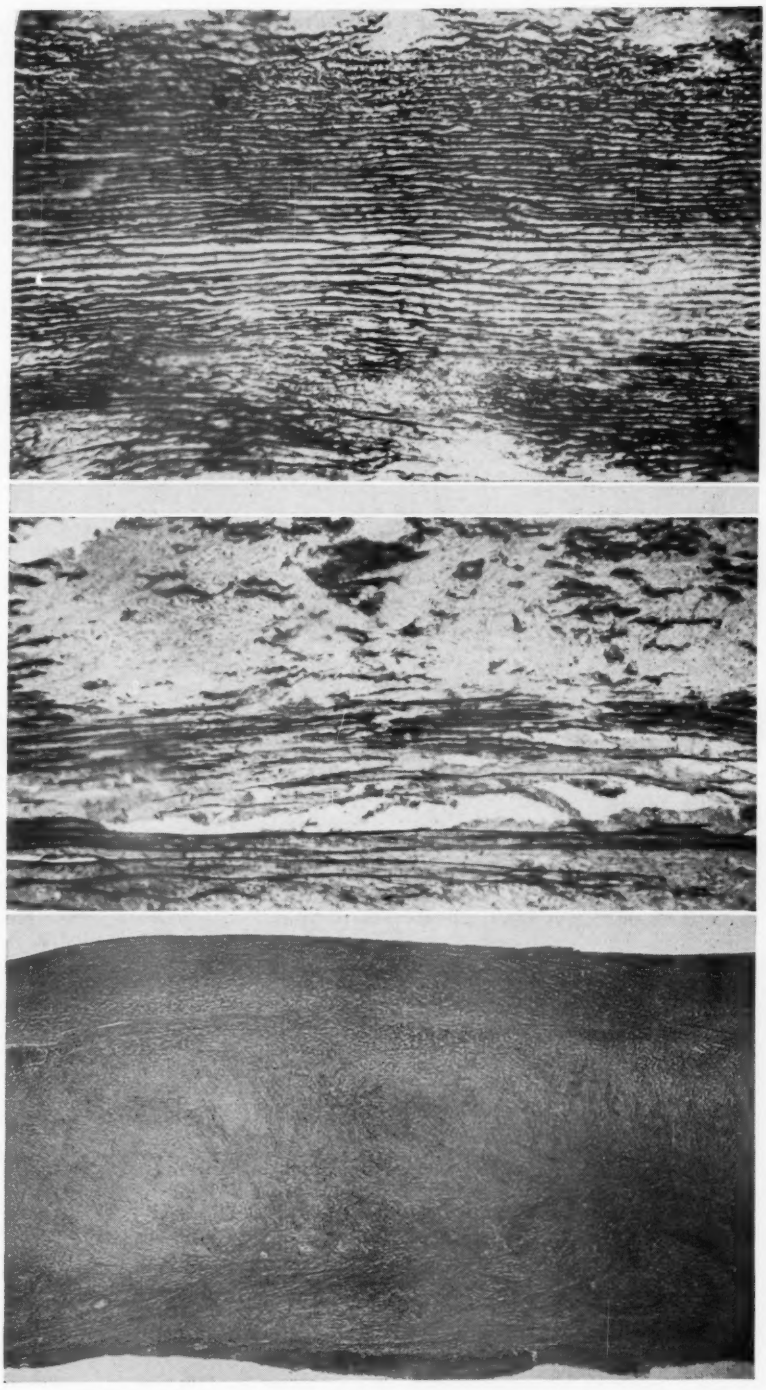

Fig. 15.—Photomicrographs of Weigert's elastic tissue stains of longitudinal celloidin sections showing the diverse microscopic fates of frozen-dried canine aortic heterografts in the thoracic aorta of growing pig. Upper panel shows fiber framework at ×80 of central portion of graft shown in lower panel of Fig. 14. Note that the fiber framework of this graft has, for the most part, retained its continuity. Middle panel shows fiber framework at ×110 of graft, 6.2 cm. long, after 197 days in aorta in which delayed fragmentation has occurred. This graft underwent neither aneurysmal change nor delayed thrombosis. However, four other grafts showing this change were thrombosed at slaughter. Lower panel shows wall at ×30 of central portion of the graft shown in the second panel of Fig. 14. The fiber framework of the original graft has disappeared, leaving only autogenous fibrous tissue.

9. *Tyrode's and Frozen-dried Canine Aortic Heterograft Composite Grafts.*—These composite grafts are entirely analogous to the Tyrode's and frozen-dried aortic homograft composite grafts. Of 11 young pigs receiving such grafts in their thoracic aorta, 2 developed aneurysms in both components of the composite graft and died of rupture of the frozen-dried portion at forty-three and forty-nine days after operation, respectively. The remaining 9 pigs came to slaughter after an average of 247 days, with a weight gain averaging 703 per cent (37 to 297 pounds). Data regarding aneurysm formation and loss of fiber continuity in the 2 components of the 11 composite grafts are in Table

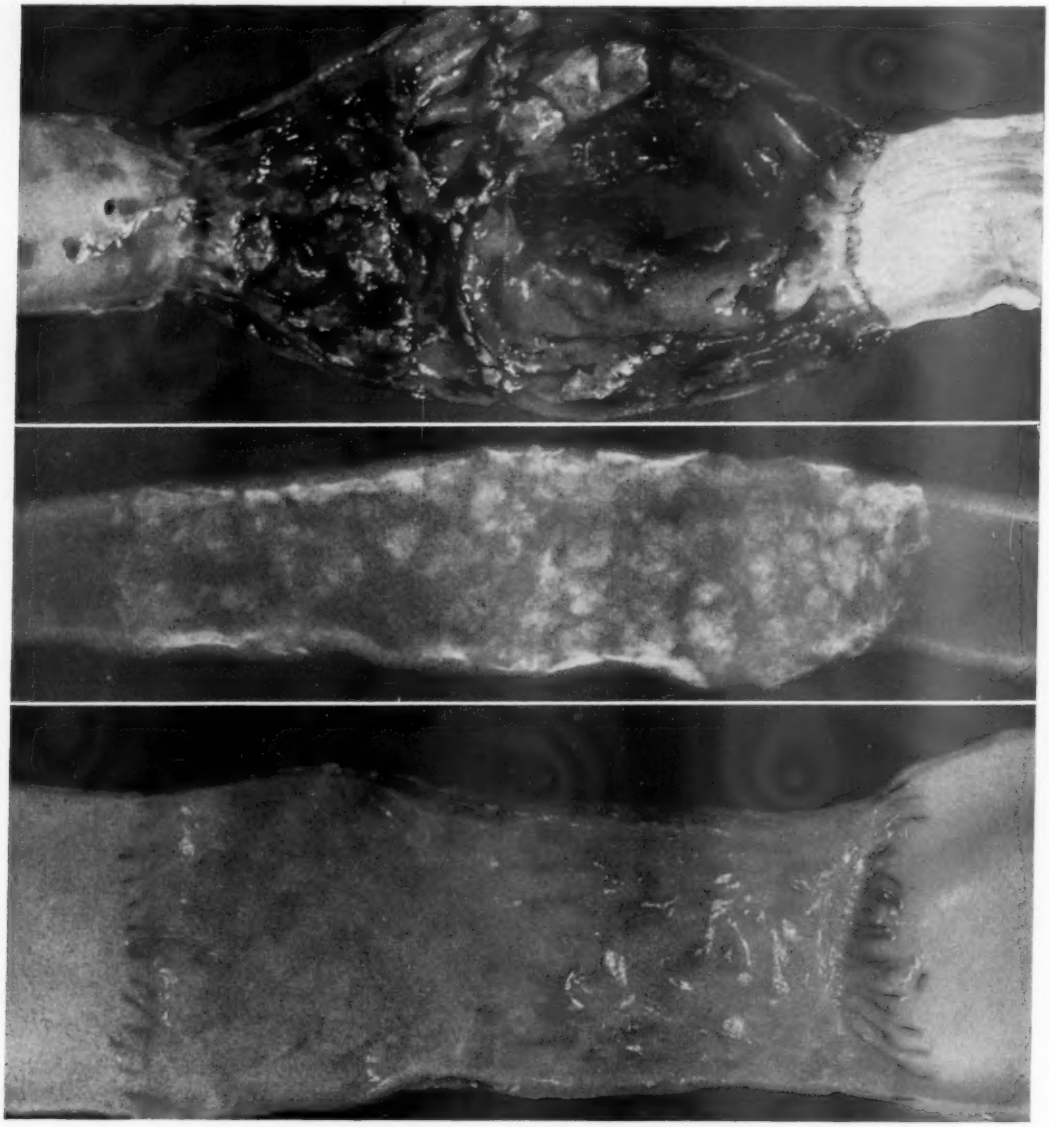

Fig. 16.—Representative Tyrode's and frozen-dried canine aortic heterograft composite grafts in thoracic aorta of growing pig. Upper panel shows photograph of open view of 11 cm. long aneurysm, the frozen-dried portion of which ruptured forty-three days after graft implantation. Middle panel shows roentgenograph of composite graft, 10.7 cm. long, after 219 days in aorta. The frozen-dried portion is to the left. Note the diffuse calcification in both components of this composite graft. Lower panel shows photograph of open view of graft, 8.3 cm. long, after 238 days in aorta. The frozen-dried portion is to the left.

IX. Freeze-dry processing did not improve the fate of the canine aortic heterograft. The gross features of 3 composite grafts are shown in Fig. 16. The microscopic features of both components of these composite grafts were the same, with loss of elastic tissue being complete or nearly so in all but 1 instance. In this respect, these grafts resembled fresh canine heterografts more than they did frozen-dried canine heterografts.

TABLE IX. TYRODE'S AND FROZEN-DRIED CANINE AORTIC HETEROGRAFT COMPOSITE GRAFTS IN THORACIC AORTA OF GROWING PIG

		ANEURYSM FORMATION IN				LOSS OF FIBER CONTINUITY IN			
	TOTAL GRAFTS	BOTH GRAFTS	NEITHER GRAFT	ONLY TYRODE'S GRAFT	ONLY FROZEN-DRIED GRAFT	BOTH GRAFTS	NEITHER GRAFT	ONLY TYRODE'S GRAFT	ONLY FROZEN-DRIED GRAFT
Number	11	2	7	1	1	10	1	0	0
Per cent of total	100	18	64	9	9	91	9	0	0

10. *Fresh Aortic Homograft–Canine Aortic Heterograft Composite Grafts.*—Three centimeter length fresh aortic homograft and canine aortic heterograft segments were anastomosed to form standard 6 cm. length composite grafts, with the position of the segments being alternated in successive grafts. Of 11 young pigs receiving such grafts in their thoracic aorta, 6 died of heterograft aneurysms (5 of rupture and 1 of inanition due to esophageal compression) an average of thirty-two days after operation, and 5 came to slaughter after

Fig. 17.—Photograph of representative fresh aortic homograft–canine aortic heterograft composite graft inflated with air to 120 mm. Hg after 255 days in thoracic aorta of growing pig. The heterograft component is to the right. Its aneurysmal character contrasts with the stability of the homograft wall.

an average of 229 days with a weight gain averaging 856 per cent (32 to 306 pounds). Data regarding aneurysm formation and loss of fiber continuity in the two components of the 11 composite grafts are in Table X. The absence of aneurysm formation in the homografts contrasts with its 73 per cent incidence in the heterografts. Also, the maintenance of fiber continuity in all

homografts contrasts with its loss in all heterografts. Two of the 5 grafts coming to slaughter showed aneurysms in their heterograft components. One of these grafts is shown in Fig. 17. The histologic findings in these 11 composite grafts were the same as those previously described for their 2 components.

TABLE X. FRESH AORTIC HOMOGRAFT–CANINE AORTIC HETEROGRAFT COMPOSITE GRAFTS IN THORACIC AORTA OF GROWING PIG

		ANEURYSM FORMATION IN				LOSS OF FIBER CONTINUITY IN			
	TOTAL GRAFTS	BOTH GRAFTS	NEITHER GRAFT	ONLY HOMO-GRAFT	ONLY HETERO-GRAFT	BOTH GRAFTS	NEITHER GRAFT	ONLY HOMO-GRAFT	ONLY HETERO-GRAFT
Number	11	0	3	0	8	0	0	0	11
Per cent of total	100	0	27	0	73	0	0	0	100

11. *Frozen-dried Aortic Homograft–Canine Aortic Heterograft Composite Grafts.*—Three centimeter length fresh aortic homograft and canine aortic heterograft segments were frozen-dried by the previously described technique, stored an average of thirty-seven days, then hydrated in saline solution and anastomosed to form standard 6 cm. length composite grafts, with the position of the segments being alternated in successive grafts. Of 12 young pigs receiving such grafts in their thoracic aorta, 5 came to slaughter an average of 242 days after operation, with weight gain averaging 771 per cent (34 to 296 pounds), and 7 died spontaneously after an average of 105 days. Data regarding aneurysm formation and loss of fiber continuity in the 2 components

TABLE XI. FROZEN-DRIED AORTIC HOMOGRAFT–CANINE AORTIC HETEROGRAFT COMPOSITE GRAFTS IN THORACIC AORTA OF GROWING PIG

		ANEURYSM FORMATION IN				LOSS OF FIBER CONTINUITY IN			
	TOTAL GRAFTS	BOTH GRAFTS	NEITHER GRAFT	ONLY HOMO-GRAFT	ONLY HETERO-GRAFT	BOTH GRAFTS	NEITHER GRAFT	ONLY HOMO-GRAFT	ONLY HETERO-GRAFT
Number	12	0	7	0	5	0	2	0	10
Per cent of total	100	0	58	0	42	0	17	0	83

of the 12 composite grafts are in Table XI. Five of the 7 pigs that died spontaneously had heterograft aneurysms (4 ruptured and 1 obstructed, Fig. 18), while none of the 5 that were slaughtered had aneurysms, but 2 were thrombosed. The absence of aneurysm formation in the homografts contrasts with its 42 per cent incidence in the heterografts. Also, the maintenance of fiber continuity in all the homografts contrasts with its loss in 83 per cent of the heterografts. The histologic findings in these 12 composite grafts were the same as those previously described for their 2 components.

12. *Frozen-dried Human Arterial Heterografts.*—Good frozen-dried human arterial segments were withdrawn from our clinical bank and used as heterografts to bridge aortic defects. These segments had been procured, prepared, and processed in the manner previously described for the frozen-dried porcine and canine aortic segments. These human arterial heterografts had been

TABLE XII. FROZEN-DRIED HUMAN ARTERIAL HETEROGRAFTS IN THORACIC AORTA OF GROWING PIG

	TOTAL GRAFTS	PIGS DYING OF RUPTURED ANEURYSM	PIGS COMING TO SLAUGHTER WITH			LOSS OF FIBER CONTINUITY
			GRAFTS OPEN		GRAFTS CLOSED	
			WITH ANEURYSM	WITHOUT ANEURYSM		
Number	12	3	3	5	1	12
Per cent of total	100	25	25	42	8	100

stored for an average period of forty-one days and varied in length from 2 to 6 cm. Of 12 young pigs receiving such grafts in their thoracic aorta, 3 died of ruptured aneurysms and 9 came to slaughter after an average period of thirty-nine and 241 days, respectively. Data of these grafts are in Table XII. The average weight gain (36 to 307 pounds) of the slaughtered pigs was 753 per cent. The gross morphologic fates of these grafts varied greatly, as shown in Fig. 19.

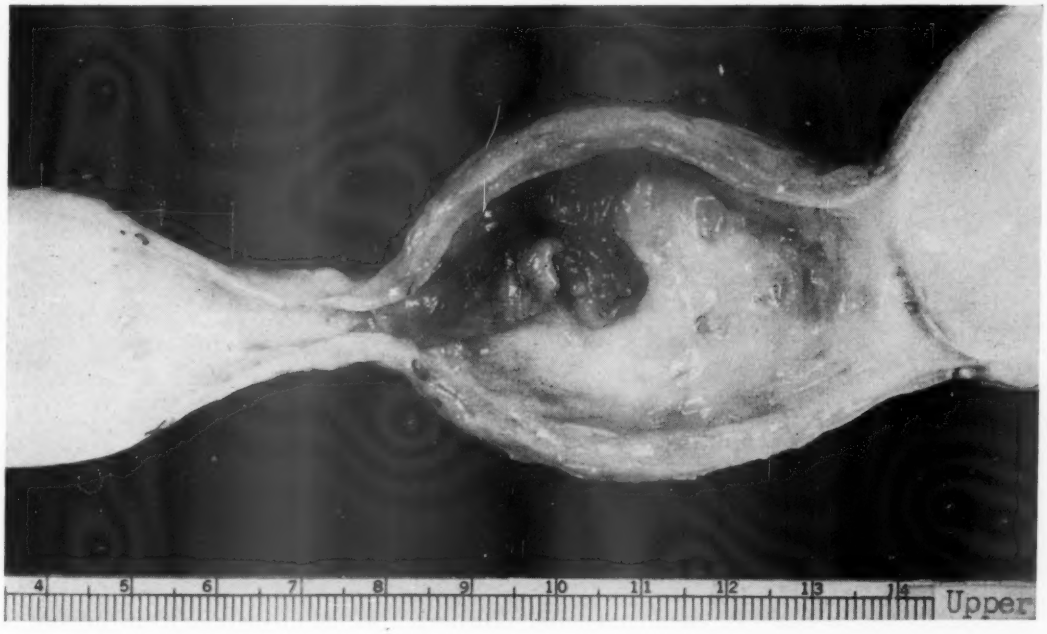

Fig. 18.—Photograph of open view of frozen-dried aortic homograft–canine aortic heterograft composite graft in thoracic aorta of growing pig for 254 days. The heterograft component is to the right. The pig died spontaneously of cardiac failure subsequent to obstruction of blood flow through the intergraft anastomosis by the ball valve action of the pedunculated thrombus arising from the wall of the small heterograft aneurysm.

The histologic findings were similar in all grafts with, as shown in Fig. 20, loss of fiber framework continuity and replacement by autogenous fibrous tissue being characteristic. This healing pattern is the same as that observed in the fresh and frozen-dried canine aortic heterografts.

Our results indicate that frozen-dried human arterial heterografts in the pig thoracic aorta are unreliable, as 6 (50 per cent) formed aneurysms and 3 of these ruptured. Of the 3 aneurysms coming to slaughter, 2 had deeply eroded into adjacent vertebrae. Frozen-dried porcine arterial heterografts

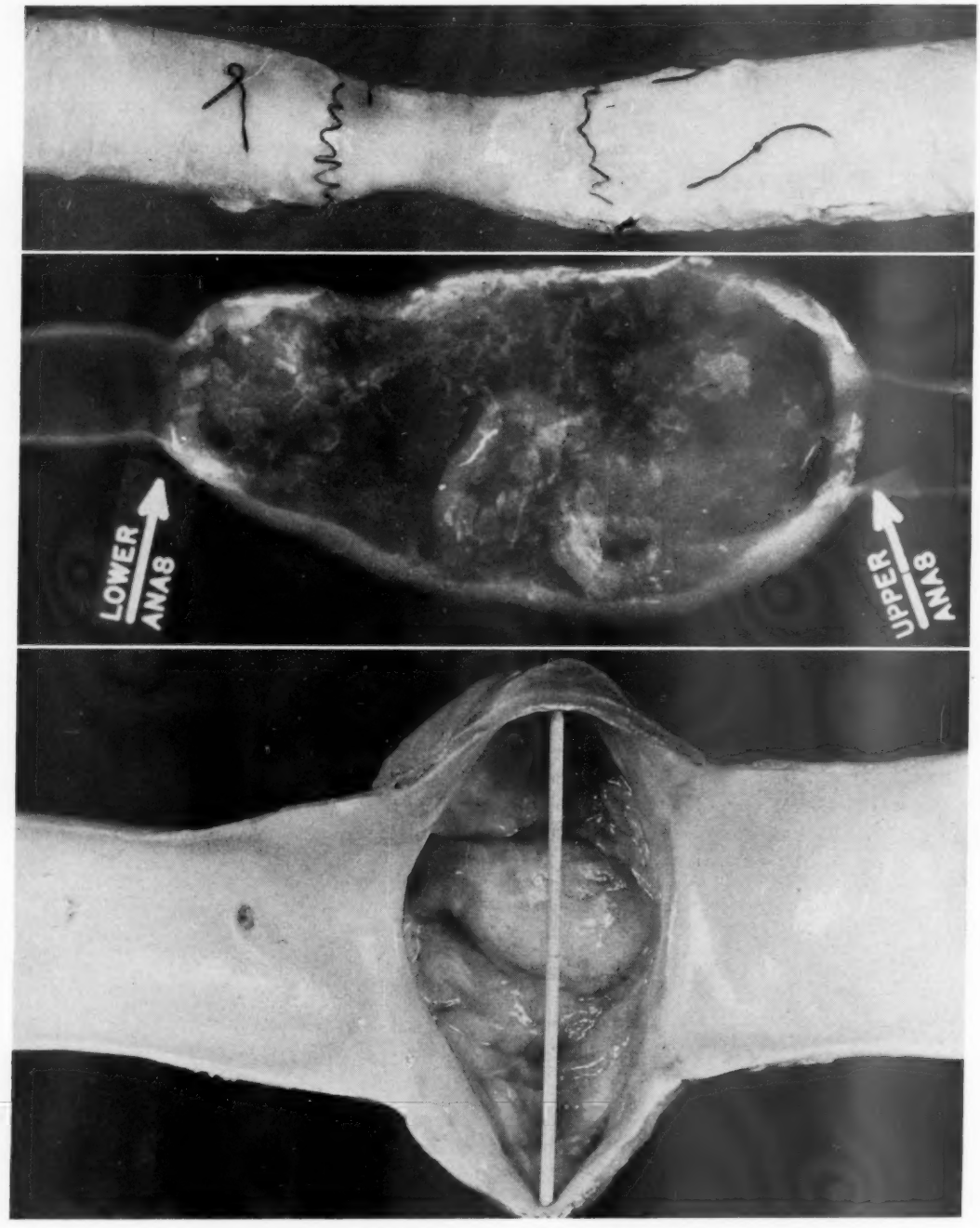

Fig. 19.—Representative frozen-dried human arterial heterografts in thoracic aorta of growing pig. Upper panel shows photograph of a graft, 3.60 cm. long, inflated with air to 120 mm. Hg after 210 days in aorta. Despite loss of fiber framework of the original graft, the autogenous fibrous tissue wall was sufficiently strong to resist successfully the dilative effect of the blood pressure. Middle panel shows roentgenograph of an aneurysm measuring 12 by 6.3 cm. which developed in 232 days from a graft measuring 5 by 0.9 cm. This aneurysm had eroded deeply into several thoracic vertebrae. Its autogenous fibrous tissue wall, in the absence of the fiber framework of the original graft, was not able to resist the dilative effect of the intraluminal pressure. Lower panel shows photograph of open view of an aneurysm measuring 5.7 by 5.6 cm. which developed in 230 days from a graft measuring 2.0 by 1.3 cm. This aneurysm, like the preceding one, had eroded deeply into the adjacent thoracic vertebrae. Note the smooth, shiny character of its inner lining.

in the human aorta would probably have similar fates. The observations of Hufnagel, Rabil, and Reed (1954) regarding ethylene oxide-treated frozen-dried porcine arterial heterografts in the peripheral arteries of man do not of necessity pertain to the aorta. Wesolowski and Sauvage (1955) observed that the fates of fresh and ethylene oxide–treated frozen-dried porcine aortic heterografts were similar in both the thoracic and abdominal aorta of the dog. Creech and associaes (1954) have observed both frozen-dried and formalinized porcine arterial segments to be unreliable grafts in the dog's arterial system.

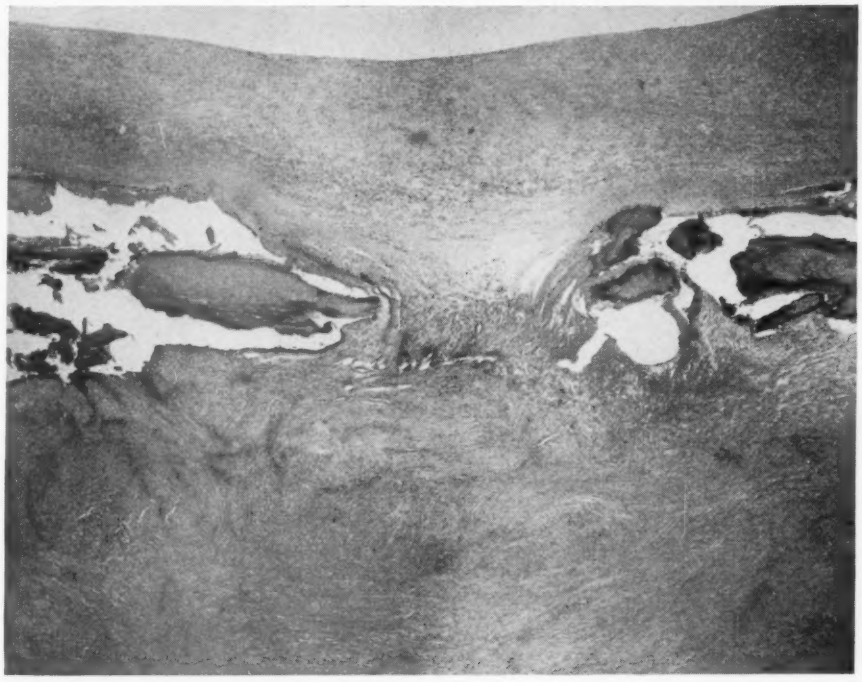

Fig. 20.—Photomicrograph at ×40 of hematoxylin and eosin stain of longitudinal celloidin section of the aneurysm shown in the lower panel of Fig. 19. The disintegration of the fiber framework of the original graft with ingrowth of autogenous fibrous elements through the defect to form an inner layer is apparent.

13. *Orlon Cloth Studies.*—Single (overlap) seam tubes of heavy weight (drapery-type) 100 per cent Orlon cloth,* having a mesh size of 55 by 90 microns, were hand-sewn by the technique shown in Fig. 21, while single- and double- (lateral) seam tubes, and patches of lightweight (lining-type) 100 per cent Orlon cloth** having a similar mesh size were machine-sewn. The hand-sewn tubes were sterilized by eighteen hours' immersion in a 1:1,000 solution of aqueous Zephiran, while the machine-sewn tubes and patches were sterilized by boiling. The hand-sewn tubes were implanted into defects (12) of the thoracic aorta of 12 young pigs, while the machine-sewn prostheses were im-

*Cloth made, up of twisted strands having thread width of 170 microns per warp and 350 microns per filter.

**Cloth made up of twisted strands having thread width of 200 microns per warp and 250 microns per filter.

planted into defects (10) of the aortic arch (2), abdominal aorta (3), abdominal wall (2), and left diaphragm (3) of 6 mature dogs. The aortic grafts in the chest were implanted with a continuous whip stitch of 5-0 silk, the aortic grafts in the abdomen by interrupted everting mattress stitches of 5-0 silk, and the abdominal wall and diaphragmatic grafts with interrupted stitches of 0 silk. Excessive blood loss through the mesh of the Orlon tubes implanted in the aorta was prevented by removing the lower clamp just long enough to trap blood within the graft and then reapplying this clamp for one or two minutes. With removal of both clamps at the end of this period, oozing of blood through the mesh of the cloth either did not occur or was small in amount and easily controllable.

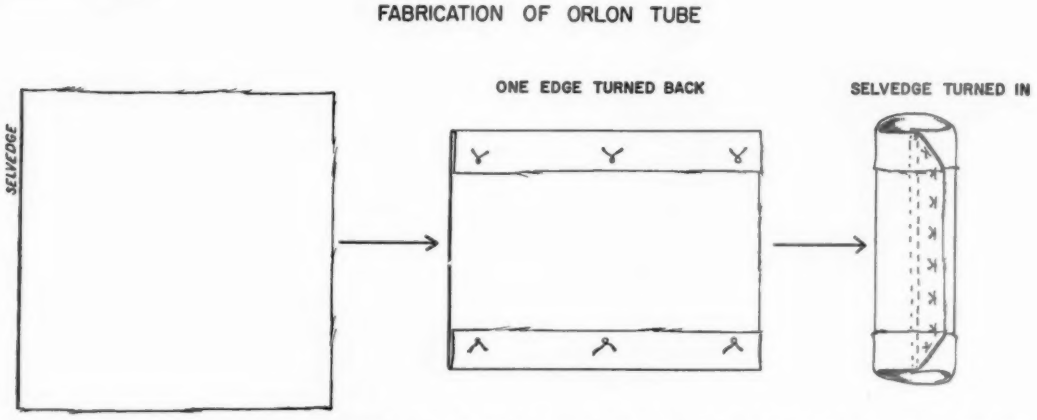

Fig. 21.—Method used for fabrication of Orlon tubes implanted into thoracic aorta of growing pig.

Of the 12 young pigs receiving single-seam Orlon tubes in the thoracic aorta, 1 died with stenosis of the distal graft 154 days after implantation and 11 came to slaughter an average of 188 days after implantation. Data of these grafts are in Table XIII. Note that the Orlon cloth developed transverse wrinkles and that calcification occurred along its inner surface in all grafts.

TABLE XIII. ORLON TUBES IN THORACIC AORTA OF GROWING PIG

	TOTAL GRAFTS	TRANSVERSE WRINKLING AND SHORTENING OF GRAFT	CALCIFICATION ALONG INNER ASPECTS OF ORLON	GOOD RESULT	STENOSIS	OCCLUSION
Number	12	12	12	6	4	2
Per cent of total	100	100	100	50	33	17

The gross features of 3 grafts are shown in Fig. 22. The 11 pigs coming to slaughter increased in weight (45 to 246 pounds), aortic length, aortic diameter, and crown-rump length an average of 463, 65, 78, and 60 per cent, respectively, while their Orlon grafts decreased in length an average of 13 per cent and in lumen diameter to the extent of double the thickness of the layer of fibrous tissue which came to line them. The thickness of this inner layer varied from 0.5 to 2.0 mm. in different grafts.

The histologic findings (Fig. 23) were similar in all grafts, with the ingrowth of fibrous elements of the outer layer through the mesh of the Orlon

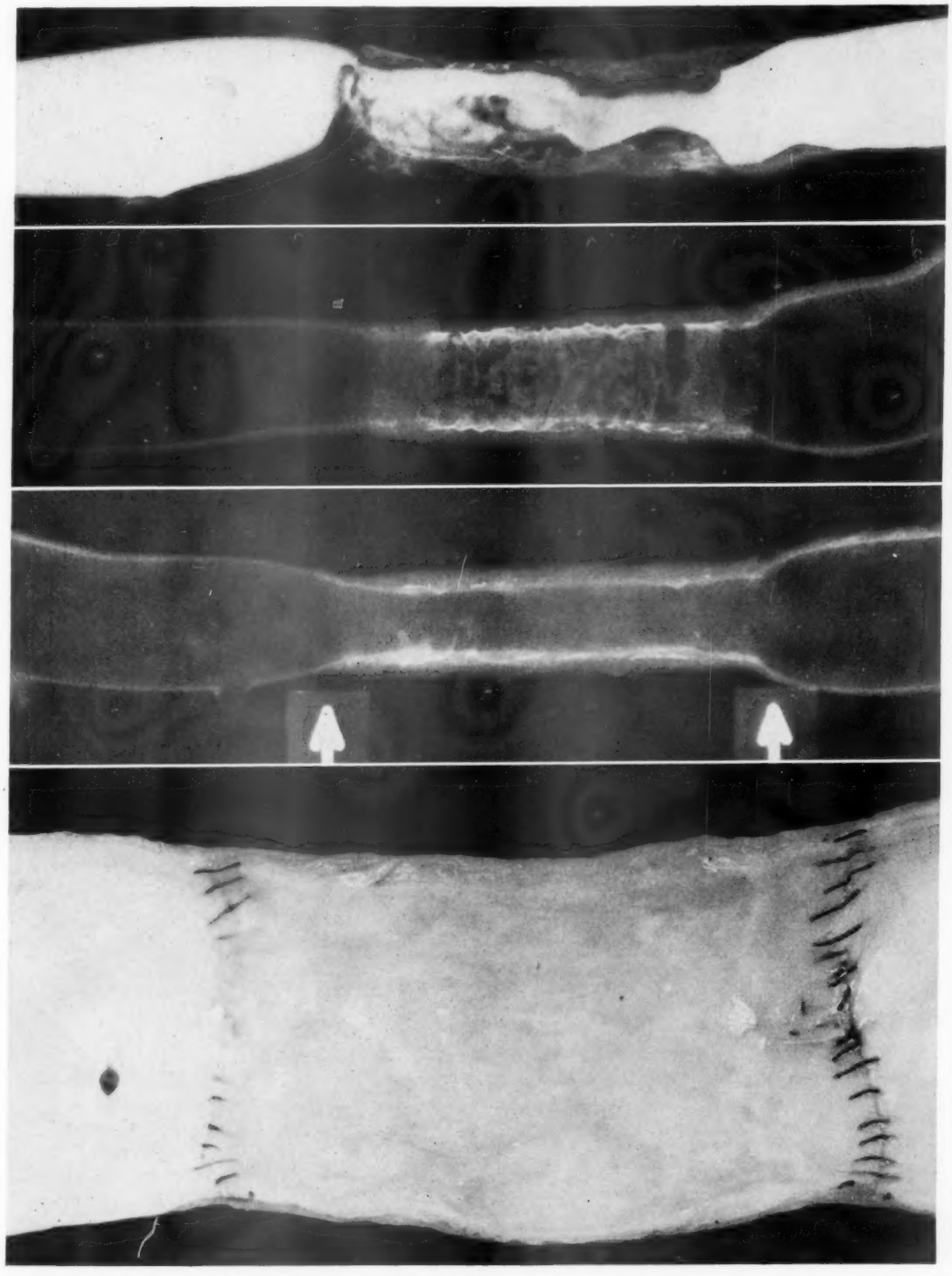

Fig. 22.—Representative 6 cm. length Orlon tubes in thoracic aorta of growing pig. Upper panel shows roentgenograph of a tube inflated with Diodrast after 203 days in aorta. Note its tortuous, attenuated lumen and the severe wrinkling of the cloth, as outlined by the calcification along its inner surface. Second panel shows roentgenograph of a tube inflated with air to 120 mm. Hg after 179 days in aorta. Note the degree of calcification present along the inner surface of the Orlon. Third panel shows roentgenograph of a tube inflated with air to 120 mm. Hg after 204 days in aorta. Little calcification has occurred along the inner surface of the cloth of this tube. Lower panel shows photograph of open view of this same tube. Note the smooth, shiny character of its inner surface.

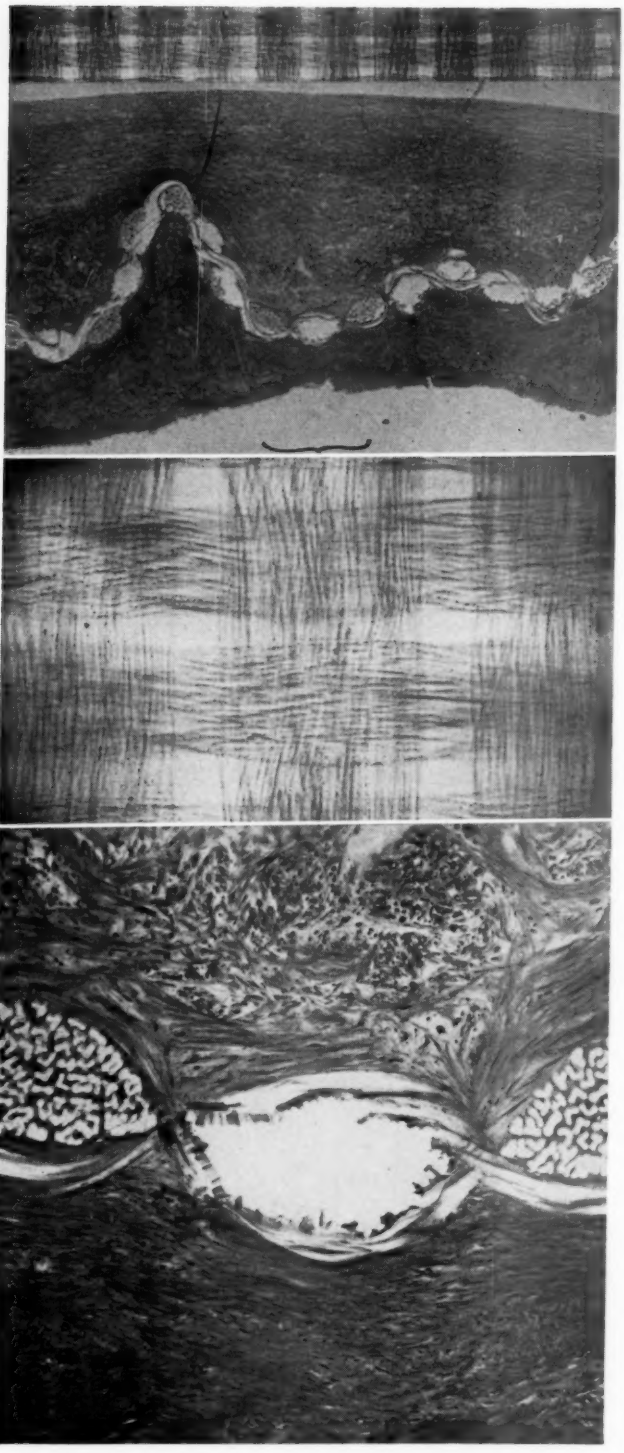

Fig. 23.—Photomicrographs of surface of longitudinal section of Orlon cloth as it lay in the tubes and of hematoxylin and eosin stain of longitudinal celloidin section of wall of Orlon tube after 137 days in thoracic aorta of pig growing from 40 to 290 pounds. Upper panel shows surface of cloth at ×25. (reduced 1/7). Second panel shows wall of Orlon tube at the same magnification and reduction. Note the wrinkling of the cloth and the ingrowth through its mesh. Third panel shows surface of Orlon cloth at ×125 (reduced 1/7). Lower panel shows region of tube wall delineated in second panel at ×125 (reduced 1/7). The growth of fibrous tissue through the mesh is apparent. Note the similarity of fiber and mesh size in panels 3 and 4.

cloth to form a thick, grayish white inner layer being characteristic. The degree of wrinkling of the cloth and extent of calcification along its inner surface varied from slight to severe in different grafts. Foreign-body giant-cell reaction was not seen, and its absence is considered to reflect the inertness of the Orlon cloth employed in these studies.

No wrinkles were present in the Orlon tubes at implantation, as they were placed under considerable tension. The reasons for the development of wrinkles in and calcification adjacent to the Orlon cloth are not apparent. There was no actual shortening of the cloth for, when removed from the fibrous tissue wall, its original length was easily regained by ironing out the wrinkles. This suggests that contracture of the fibrous tissue wall was responsible for the wrinkling. If this be so, whether or not such contracture was stimulated by the Orlon is a matter for conjecture. The calcification along the inner surface of the Orlon suggests that the nutrition in this region was deficient. The absence of aneurysm formation in all grafts despite severe stress over comparatively long periods attests to the strength and stability of the Orlon fiber framework. The tensile strength of the Orlon was, by gross testing, the same at slaughter as at implantation.

Wesolowski and Sauvage (1955) have observed that straight tubes prepared from the heavy-type Orlon cloth functioned satisfactorily in the dog's thoracic aorta, while Y tubes of this same cloth used to replace the aortic bifurcation did not, due to delayed "wrinkle" obstruction. Curves, branches, small caliber, or use in arteries subjected to angulation increase the possibility of failure of Orlon mesh prostheses due to thrombotic or "wrinkle"-type obstruction. The problem of kinking was clearly demonstrated in each of 2 normothermic dogs, whose entire aortic arch was resected and replaced with a lightweight Orlon mesh prosthesis. One dog survived twenty minutes and the other forty-eight hours after graft implantation before dying of cardiac failure secondary to graft obstruction by kinking in regions of angulation.

The grafts in the thoracic aorta of the pig were implanted using a continuous whip stitch in order to decrease the duration of circulatory interruption. Subsequent autopsy examination revealed that end-on coaptation had not been uniformly achieved, as the Orlon had occasionally invaginated into the aorta. At the upper anastomosis this led to the formation of blind pockets extending along the outer aspect of 3 of the tubes. Subsequently, we bridged defects of the abdominal aorta of 3 dogs with 6 cm. length lightweight Orlon tubes that were joined to the aorta with interrupted everting mattress sutures of 5-0 silk. Although taking longer than the whip stitch, excellent approximation and subsequent healing were attained. Accordingly, we advise that the upper anastomosis should be everted when time, exposure, and the condition of the recipient artery render this feasible. We found the lightweight Orlon tubes utilized in the canine studies easier to suture than the heavier tubes employed in the porcine experiments. Thus, for technical reasons we recommend that tubes should be fabricated from the lighter weights of Orlon cloth.

Two full-thickness (excluding skin) defects, over 100 sq. cm. in size, of the abdominal wall and 3 similar-sized defects of the left diaphragm were

surgically created in dogs and then bridged with patches consisting of a single layer of lightweight Orlon cloth with cuffed-back edges. The 3 dogs with the diaphragmatic patches were sacrificed at two, four, and eight weeks, respectively, while the 2 dogs with the abdominal wall patches were sacrificed at four and seventeen weeks after operation, respectively. The repair sites were sound in each instance. The Orlon patches had remained attached to the wound edges despite the severe stress to which they had been subjected. None of these 5 wounds showed evidence of infection. The abdominal wall Orlon developed many wrinkles despite being placed under extreme tension and became covered externally by a thick layer of fibrous tissue that was continuous through the mesh with a thin inner layer of fibrous tissue to which omentum or loops of bowel were adherent. The diaphragmatic Orlon developed few wrinkles and rapidly became enveloped by fibrous tissue that was continuous through the mesh of the cloth (Fig. 24). The entire inferior surface of each of the 3 patches was adherent to the underlying stomach and liver, in contrast to the superior surface, which was free over its entire area except for a small region of adherence to the lung in 1 instance. Thus, the serosa of the stomach and liver appears to have been the main source from which fibrous elements proliferated to envelop the diaphragmatic patches. Each patch looked excellent at sacrifice, even the two-week specimen being completely enveloped by fibrous tissue. This rapidity of healing and excellence of results suggest that the stomach and liver are ideal beds upon which to lay such a prosthesis.

With respect to the usefulness of Orlon fabric as a prosthesis in the repair of musculofascial defects, the observations of Sanford, Riley, and Sauvage (1955) seem pertinent. These investigators observed Orlon fabric to predispose to the development of chronic infection following standardized contamination of the wound, while Tantalum mesh, employed under similar conditions did not. Thus, Orlon fabric appears inferior to Tantalum mesh as a prosthesis for repair of musculofascial defects in the presence of wound contamination.

We (1955) have observed tubes of heavy-type Orlon cloth implanted in the thoracic inferior vena cava of the growing pig to become either severely contracted or completely obliterated. This complication is probably due to the inability of the venous pressure to prevent progressive contracture of the fibrous tissue encasement of the Orlon tube from occurring. This suggests that Orlon cloth prostheses are suitable for use only in the high-pressure arterial circuit or in areas where contracture of the prosthesis will not be harmful, as in musculofascial defects.

In summary, our studies of Orlon prostheses in different biologic environments suggest that (1) these prostheses are well tolerated by the host, (2) fibrous elements of the surrounding tissues proliferate to encapsulate the prostheses by an outer layer from which ingrowth through the interstices occurs to form an inner layer, and (3) the pattern of healing leading to the fibrous tissue envelopment of Orlon tubes employed to span aortic gaps appears to be the same as that of Orlon patches employed to bridge musculofascial defects.

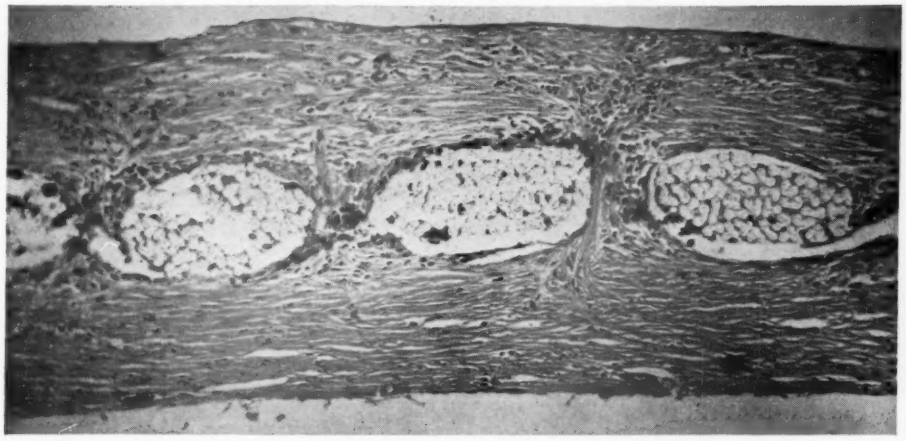

Fig. 24.—Photomicrograph at ×100 of hematoxylin and eosin stain of paraffin section of Orlon patch twenty-eight days after use as graft to bridge canine diaphragmatic defect. The stomach was adherent to the inferior surface of this section, while the superior surface was free. Note the ingrowth of fibrous tissue through the mesh of the cloth.

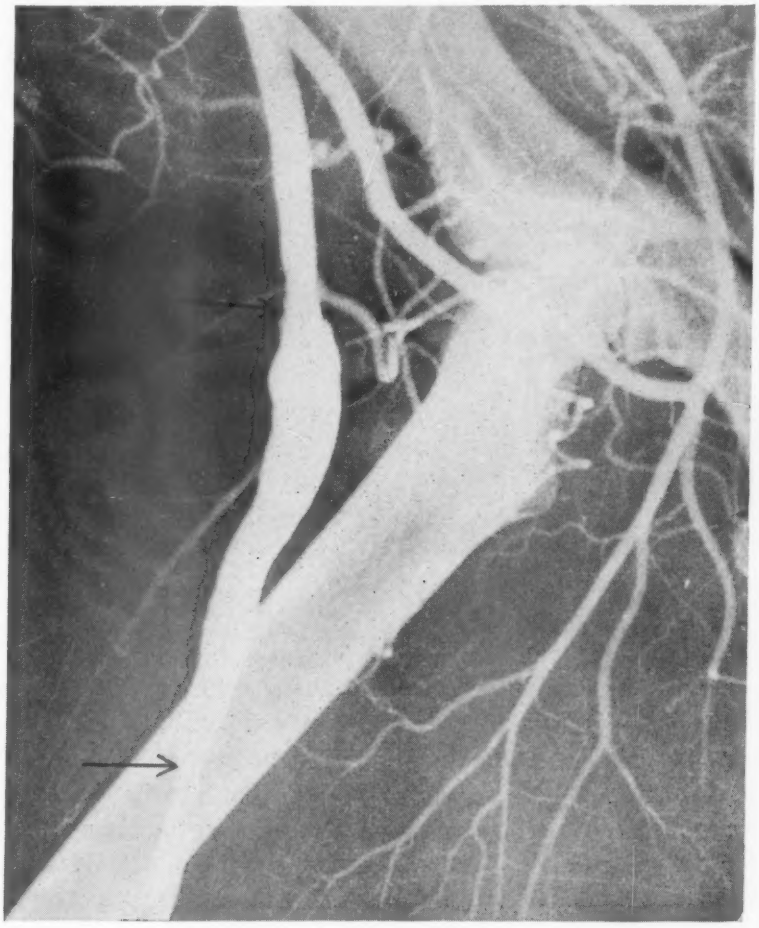

Fig. 25.—Twelve-month arteriogram of 6 cm. length fresh venous autograft in canine femoral artery.

14. *Fresh Autogenous Femoral Vein Grafts.*—Six centimeter length segments of the femoral vein were resected, reversed, and used to bridge defects of the concomitant femoral artery. Of 8 mature dogs receiving such grafts, 7 remained patent. These latter were observed for an average period of one year, during which time moderate, but not progressive, dilatation of 4 occurred as revealed by serial arteriograms at six and twelve months. The twelve-month arteriogram of a graft is shown in Fig. 25. At sacrifice these 7 grafts were thickened and tightly bound to and supported by the surrounding musculofascial tissues. However, their valves were intact, thin, mobile, and histologically normal. These findings (Fig. 26) indicate that the autogenous vein wall persisted as a viable, anatomically intact structure.

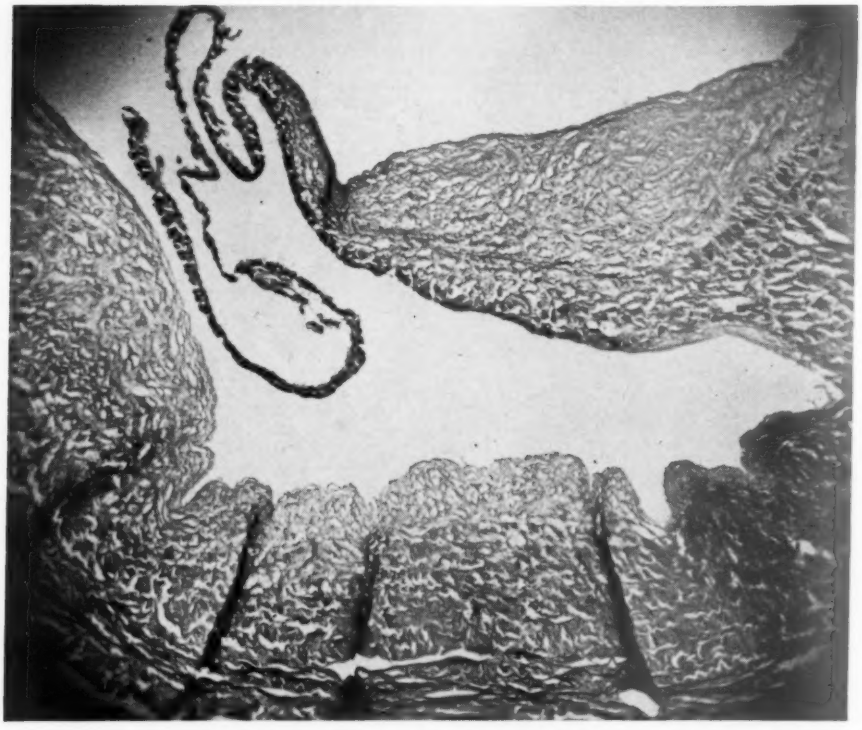

Fig. 26.—Photomicrograph at ×100 of Weigert's elastic tissue stain of longitudinal paraffn section across valve site of fresh venous autograft in canine femoral artery for 382 days. Note that the valve has remained a thin, anatomically intact structure.

The degree of dilatation observed in these grafts was not consequential. Hughes and Jahnke (1954), of the Walter Reed Army Medical Center, have not observed autogenous saphenous vein grafts in major peripheral arteries to dilate in a series of 40 patients followed from one to four years. These data confirm the dictum that aneurysmal dilatation of autogenous veins implanted in arteries does not occur in regions providing good musculofascial support to the grafts. Regarding the need of the military for means of blood vessel replacement in the extremities as a result of trauma, one may state that the readily available, muscular, autogenous saphenous vein answers this need satisfactorily in the vast majority of instances.

15. *Healing of Homografts and Host Aorta Following Removal of Inner Wall.*—The healing of the inner wall of 6 frozen-dried (average age of 191 days) and 2 fresh aortic homografts was studied by the following method in 6 dogs. Prior to implantation, these 8 homograft segments were turned inside out and the inner third of their wall removed to within 5 mm. of the ends, in one half as a 5 mm. wide strip and in the other half as a complete circumferential sheet. Then, these segments were turned outside in and implanted as single homografts, 9 cm. in length, into the thoracic aorta of each of four dogs and as composite homografts (fresh and frozen-dried), 15 cm. in length, into the thoracic aorta of each of 2 other dogs. All dogs survived operation and subsequently were sacrificed after an average period of 121 days. All grafts were patent, although extensive mural thrombosis was present in 3. Microscopically, the defect in the homograft wall was not healed unless autogenous fibrous elements were able to make their way to the inner surface.

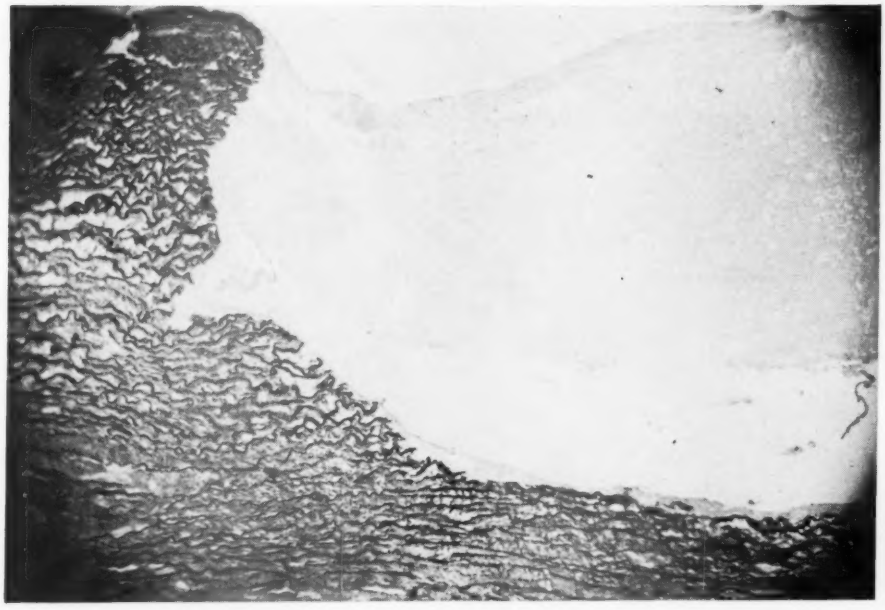

Fig. 27.—Photomicrograph at ×80 of Weigert's stain of longitudinal parffin section across upper end of stripped-out zone of frozen-dried aortic homograft after 183 days in canine thoracic aorta. Note the trailing thrombus and the complete absence of cellular proliferation from the homograft wall.

This occurred in 1 of the 2 fresh homografts and in 1 of the 6 frozen-dried homografts. Lack of formation of an inner layer at the site of inner wall removal in a frozen-dried graft is shown in Fig. 27.

The healing of the inner wall of the autogenous canine thoracic aorta was studied by the following method in 3 dogs. The thoracic aorta was freed, opened longitudinally, stripped completely of the inner third of its wall for an average distance of 4 cm., and then closed with a continuous whipstitch. These dogs were then sacrificed at intervals of two, four, and eight weeks after operation, respectively. In each instance the stripped segment was free of

fibrin and covered in most areas by glistening, smooth, translucent tissue that appeared to have origin from the contiguous underlying wall. These gross and microscopic findings are shown in Fig. 28. It is worthy of comment that this reformed inner layer was thinnest near the orifices of the ligated intercostal branches, even though the intimal surface in the depth of these recesses was not removed by the stripping procedure. This and the absence of sheet-like ingrowth from the margins of the stripped-out zone strongly suggest that

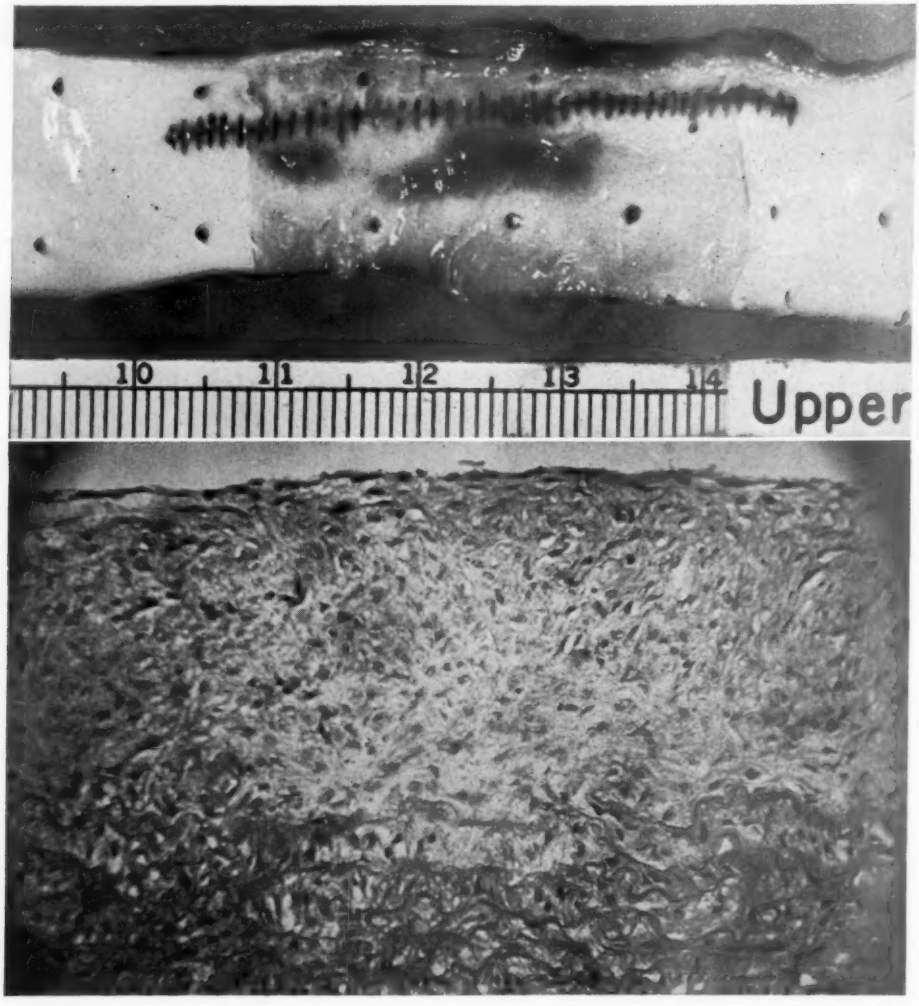

Fig. 28.—Segment of host (canine) thoracic aorta fourteen days after removal of its inner wall. Upper panel shows photograph of opened segment revealing the arteriotomy line to be well healed and the surface of the stripped-out zone to be free of fibrin and comparatively smooth. Lower panel shows photomicrograph at ×180 of hematoxylin and eosin stain of longitudinal section of wall from central portion of the stripped zone. Note the layer of fibrous tissue which has rapidly formed to resurface the denuded wall of the host aorta.

the new inner layer was the product of fibrous proliferation by the underlying wall, with each area being responsible for itself in this respect. The findings presented in this and the other fourteen sections of this paper indicate that the healing of the arterial wall is a function of the contiguous ar-

terial or periarterial tissues and not of the blood flowing through its lumen. In brief, gaps or defects in the arterial wall are sealed by fibrin and then healed by ingrowth of fibrous elements from the surrounding tissues. Thromboendarterectomy wounds probably heal by migration to the inner surface and proliferation there of mural fibroblasts. Thus, thromboendarterectomy, if properly performed, appears compatible with sound healing and good function.

DISCUSSION

Host-graft junction areas are healed by ingrowth of fibrous elements from both the contiguous arterial and periarterial tissues, while junction areas between nonviable grafts are healed by ingrowth of fibrous elements of only the periarterial tissues. This healing pattern is basically the same as that manifest by the body in other areas where specialized elements are not regenerated.

We have observed the same pattern of healing in all the grafts reported in this paper; namely, that of fibrous ingrowth from the arterial and periarterial tissues. The location, degree, and extent of this ingrowth into the graft are determined by the fate of the cells and fibers of the original graft. If cellular viability of an arterial graft is lost, the inner portion of its fiber framework undergoes a spatial rearrangement into an acellular, condensed mass of elastic tissue. Thus, the fibrous ingrowth from the periarterial tissues cannot make its way to the inner surface of this impenetrable barrier except by lateral spread from the stock of fibrous tissue growing in through the anastomotic junction site. The extent of this lateral spread from the anastomosis is limited and rarely reaches the center of grafts as long as 6 cm. If the acellular, condensed mass of elastic tissue loses its continuity and becomes pervious, the fibrous elements of the periarterial tissues grow through and form an inner layer on its surface. The strength of the graft wall is, for a considerable period, dependent largely upon the maintenance of the continuity of its fiber framework. If this continuity be lost, the time at which such loss occurs is of the greatest importance in determining the gross morphologic fate of the graft. Should early loss occur, rupture ensues. If continuity persists longer, the autogenous fibrous ingrowth may have attained sufficient strength to resist frank rupture but not aneurysmal dilatation. However, if fiber continuity persists until the autogenous ingrowth is of sufficient strength to resist the dilative effect of the intraluminal pressure, a stable wall will then remain despite the subsequent loss of fiber framework continuity.

Calcific changes occur in and about the condensed, acellular masses of elastic tissue. Such changes of themselves do not impair the ability of the graft to serve as an effective conduit. However, these changes must increase the liability of the graft to delayed thrombosis, dissecting aneurysm formation, or actual rupture. Accordingly, their development is to be viewed with concern.

An intact fiber framework in an arterial graft that has lost its cellular viability prevents significant dimensional change either of a passive nature in response to the dilative effect of intraluminal pressure or of an active nature in reponse to growth stimulation.

Grafts are needed for the correction of arterial defects not manageable by end-to-end anastomosis. The type of graft one uses is dictated by the pathologic process present and his knowledge of and experience with the available grafts. In this regard we shall consider certain aspects of the fates of arterial autografts, homografts, and heterografts and of synthetic fabric tubes that influence the clinical decisions as to which of these to employ.

Arterial Autografts.—These grafts maintain both cellular viability and fiber continuity. They remain histologically identifiable as normal arteries and have no more liability to complications than does the rest of the arterial system of the patient. They are the perfect graft. Unfortunately, their relative unavailability greatly limits their application.

Arterial Homografts.—These grafts, even though living at implantation, usually lose cellular viability but maintain fiber continuity. The fibroblasts of the adjacent host tissues proliferate and invade the homograft wall to varying depths and spread from the anastomotic site along its inner surface for short distances. Fibrous tissue from the anastomotic junction grows out to cover the inner surface of the adjacent 1 to 2 cm. of graft and 2 to 3 mm. of aorta. The extent to which the intima of the aorta contributes to this process has not been determined. Some fibrillar elements taking the elastic tissue stain extend out from the aortic intima for 1 to 2 mm. along the inner surface of the fibrous tissue at the anastomotic site. In most instances the inner portion of the homograft fiber framework becomes condensed to form an acellular, relatively inert, mass of foreign protein which is impervious to the ingrowth of host tissue and remains so for periods of at least a year. Thus, only at, and adjacent to, the anastomosis (1 to 2 cm.) and to those exceptional sites where the fiber framework happens to remain pervious to the ingrowth of host tissue, or becomes so subsequently, does a layer of host tissue form along the inner surface of the homograft fiber framework. Accordingly, most homografts longer than 3 to 4 cm. are devoid centrally of an inner lining of host tissue. In grafts of such length as to be devoid of an inner lining centrally the condensed fiber framework of the original graft serves as an effective conduit for the passage of blood. This surface of itself does not promote thrombosis. However, degenerative changes occurring in this condensed, acellular mass of elastic tissue might be expected to favor the development of delayed thrombosis.

In the early period following implantation, the strength of the graft resides in its homograft fiber framework. Subsequently, the autogenous fibrous tissue reaction about the homograft is sufficient of itself to serve as a suitable conduit of stable dimensions. It is difficult to state how long the graft is dependent upon the integrity of the homograft fiber framework for

its strength. As a generalization we suggest that loss of continuity of the homograft fiber framework before three months would favor frank rupture or aneurysm formation while such loss after twelve months would not.

In essence, arterial homografts are foreign fiber frameworks that through condensation of their inner portion become impervious to the ingrowth of fibrous elements of the periarterial tissues. Because of fiber framework resistance, these grafts are resistant to aneurysm formation, unable to respond to growth stimulation, and liable to calcification. The calcific changes are least in fresh grafts, maximal in grafts dried by evaporation, and intermediate in frozen and frozen-dried grafts. Because of their inability to respond to growth stimuli and their distinct liability to calcification, arterial homografts should not be used in growing patients unless they are the only suitable means by which a life-endangering hemodynamic abnormality may be corrected. The adult patient requiring surgical correction of arteriosclerotic pathologic processes presents a vastly different problem than does the young patient with a congenital defect. In the former case the graft probably has less liability to serious complications than does the rest of the arterial system of the patient, while the reverse is true in the latter case.

Arterial Heterografts.—These grafts, even though living at implantation, lose cellular viability and in most instances fiber continuity as well. Because of this loss of fiber continuity, these grafts, in contrast to homografts, present an unstable wall which is prone to aneurysm formation and rupture. Accordingly, they must not be used unless they are the sole means for correction of hemodynamic abnormalities of greater risk to the patient than that entailed by the use of such grafts. The disintegration of the fiber framework allows the fibrous elements growing in from the periarterial tissues to reach the inner surface and form an inner layer. The wall of a heterograft aneurysm is analogous to that of an arteriovenous fistula or a false aneurysm of any standing. Probably the healing pattern in these latter situations is the same as in the heterografts, but without the temporary presence of a foreign fiber framework. Freeze-drying seems to delay, but not to prevent, the loss of continuity of the heterograft's fiber framework. Because of this, the incidence of delayed thrombosis is higher while aneurysm formation is lower in the frozen-dried aortic heterograft than in the fresh aortic heterograft.

Synthetic Fabrics.—The pioneer work of Voorhees, Jaretzki, and Blakemore (1952) with Vinyon-N began an exciting chapter in the field of reconstructive arterial surgery. Much work by many investigators has been done since with a variety of plastic materials. At present, five synthetic fibers appear suitable for use in the arterial system. These are Vinyon-N, Nylon, Orlon, Dacron, and Teflon. These fibers have less inherent elasticity than their homograft counterpart, the elastic fiber. Accordingly, prostheses fabricated from these fibers are less adaptable than arterial homografts. Consequently, although these prostheses may be occasionally employed successfully to replace branched segments of small arteries, they are best suited for bridging straight defects of large arteries where branches, angulation, and small caliber

are not considerations. The elasticity of a fabric is influenced by its weave. For example, a braided tube of a given fiber possesses more elasticity than does a tube of taffeta weave of the same fiber. Fiber frameworks of the above-mentioned fibers are stable, relatively inert, and, following use as arterial grafts, come to be enveloped by an autogenous fibrous tissue wall, the inner portion of which is derived from the outer portion by permeation of fibrous elements through the mesh of the fabric. Hufnagel (1955) believed that failure of ingrowth of fibrous elements through the mesh to form a lining of host tissue along the inner surface of the synthetic fiber framework may lead to a high incidence of delayed thrombosis in the prosthesis. The importance of pore size to the ingrowth of fibroblasts through the mesh needs further clarification. The article of Deterling and Bhonslay (1955) is a step in this direction. Hufnagel (1955) has successfully employed synthetic fiber frameworks to replace pathologic portions of the arterial systems of 75 patients, including the aortic bifurcation in 50 instances. The majority of these prostheses have been fabricated from light weight Orlon cloth having a mesh size of 30 by 50 microns. Much still remains to be learned of the synthetic fiber frameworks suitable for arterial replacement before they can be widely employed clinically with the same comparative safety that, at present, attends the proper use of appropriate homografts. However, as of this time one may say that the correct use of suitable synthetic fiber frameworks for bridging selected arterial defects is compatible with sound healing and satisfactory function.

With respect to the surgical management of aneurysms of the major arteries in the body cavities, the choice of synthetic fiber framework prosthesis or homologous artery fiber framework prosthesis reflects more a particular interest in one framework than it does a conviction of the superiority of that framework as compared to the other. Regarding the surgical management of occlusive arterial disease of arteriosclerotic origin involving the aorta and its iliac branches, one may employ thromboendarterectomy, arterial homograft replacement, or any one of the suitable synthetic fiber frameworks that are currently available for replacement and obtain equally good results provided that the method employed be applied correctly.

The management of occlusive arterial disease of arteriosclerotic origin distal to the inguinal ligament often presents difficult problems. However, if the arterial bed at, and distal to, the bifurcation of the popliteal artery is reasonably patent, one can successfully manage these cases surgically with gratifying results, provided that sufficient attention be directed toward technical detail. The concept of by-pass grafts placed as shunts around sites of arterial obstruction utilizing end-to-side anastomoses as originally introduced by Kunlin (1951) and subsequently adopted and modified by Linton and Menendez (1955) and by Crawford and DeBakey (1955) appears to be a valuable contribution toward rendering the care of such patients feasible on a practical basis. Linton has suggested that the high incidence of delayed thrombosis (50 per cent) occurring in arterial homografts employed as replace-

ment grafts is related to the anastomotic constriction resulting from end-to-end union with the host artery. The experimental work of Anzola, Palmer, and Welch (1952) suggests that the high incidence of delayed thrombosis in long-length arterial homografts of small caliber is related to the nature of the graft and not to its method of union with the host artery. Further observation of long-length arterial homografts of relatively small caliber placed as by-pass shunts (utilizing end-to-side union without constriction) will be necessary in order to clarify this important point. The use of the autogenous saphenous vein as a by-pass graft under such circumstances likewise merits careful evaluation. Olwin (1955) and Murphy, Aust, and Felder (1955) have been favorably impressed with such grafts. Julian, Dye, Olwin, and Jordan (1952) reported good results with replacement type vein grafts in the superficial femoral artery. The follow-up of these grafts (Olwin, 1955) has revealed that of 11 venous autografts and 4 venous homografts initially successful, 9 venous autografts and 2 venous homografts have maintained patency over a 3 to 5 year period. It should be pointed out that the muscular nature of the wall of the greater saphenous vein renders it, as compared to other veins, uniquely fitted to withstand arterial pressures. The value of thromboendarterectomy distal to the inguinal ligament remains to be fully determined. Cannon and Barker (1955) reported favorably on thromboendarterectomy in the femoral artery and advocated the use of an internal stripper for this purpose. However, the extensive dissection required, the prolonged time of operation, the liability to subsequent rupture if the endarterectomy be carried too deeply, and the probability of early thrombosis if the endarterectomy be not carried deeply enough make this valuable technique undesirable under many circumstances. The proper place of the synthetic fiber frameworks in the armamentarium of peripheral arterial reconstructive surgery is in a constant state of revision. Hufnagel (1955) believes that the braided Teflon tube is the best synthetic fiber framework currently available for use in the peripheral arteries. Lewis (1955) believes that Ivalon (polyvinyl sponge) tubes, because of their elasticity, are well suited for use in the peripheral arteries. Edwards and Tapp (1955) believe that braided, crimped Nylon tubes, possessing flexibility to 180 degrees without kinking, offer promise for use in peripheral arteries. Suffice it to say that none of the plastic prostheses currently available are as adaptable as appropriate arterial homografts or venous autografts. However, the future undoubtedly will see the development of better vascular prostheses than any now available for use in the peripheral arteries.

The field of blood vessel repair and replacement is a dynamic one. The accomplishments of the past and of the present are but steps leading toward goals to be reached in the future which now seem impossible of attainment. If the results reported in this paper serve as such a step, the effort expended in their determination will have been justified.

SUMMARY

The material of this paper consists of 230 grafts implanted in 170 animals (153 young pigs and 17 mature dogs). Studies have been made in the arterial

system of the healing and fate of arterial autografts, homografts, and heterografts, of venous autografts, and of segments of the host aorta from which the inner wall has been removed; and in both the arterial and musculofascial systems of the healing and fate of Orlon cloth prostheses. On the basis of these studies we suggest that the healing of arterial grafts is the same as that of other tissues that do not regenerate specialized elements. The basic pattern of arterial graft healing consists in the ingrowth of fibrous elements from the contiguous arterial and periarterial tissues into the graft. The end results observed with different types of grafts varies widely. However, these differences are explainable on the basis of whether the cellular viability and fiber continuity of the original graft are maintained or not. The healing pattern of Orlon cloth prostheses used to span aortic gaps is the same as that of Orlon cloth prostheses used to bridge musculofascial defects. In each instance the cloth presents a stable wall which is healed by the ingrowth of fibrous elements from the surrounding tissues through its mesh. The host aorta from which the inner wall has been removed heals by migration to the inner surface and proliferation there of mural fibroblasts.

Arterial autografts were observed to be perfect grafts. Fresh venous autografts implanted in arteries receiving good support from surrounding musculofascial tissues were observed to persist as viable, anatomically intact structures which were not subject to aneurysmal dilatation. Arterial homografts were observed to be resistant to aneurysm formation, unable to respond to growth stimulation, and liable to calcification. Arterial heterografts, differing from homografts, were observed to be unstable and prone to aneurysm formation and rupture. Orlon tubes were observed to be resistant to aneurysm formation, but prone to wrinkling and liable to calcification. The denuded inner wall of the host aorta was observed to heal quickly and to have little tendency toward mural thrombosis.

To Colonel William S. Stone, MC, USA (Ret.), formerly Commandant, Army Medical Service Graduate School, now Director of Medical Education and Research, University of Maryland, we extend our sincere thanks, for without his support and helpful advice this work could not have been accomplished.

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Editorial

Vascular Substitutes

The rapidly increasing use of excisional therapy for the treatment of arterial disorders has resulted in a demand for vascular transplants that far exceeds the supply. Because of difficulties of procurement, arterial homografts are not available in sufficient number, and the predicted widespread establishment of "blood vessel banks" has not become a reality. Furthermore, to some vascular surgeons at least, the degenerative changes occurring in experimental homografts constitute a distinct disadvantage to their clinical use. As a result, a search for suitable arterial substitutes is now being conducted with an intensity that approaches the phenomenal. Following closely upon the pioneering efforts of Voorhees and Blakemore, Shumacker, and Hufnagel, investigation of vascular prostheses made from various types of synthetic materials was begun in practically every surgical laboratory. Vinyon-N, nylon, Dacron, Orlon, Ivalon sponge, silica oxide, polyethylene, and methyl methacrylate are among the materials being studied. The prostheses are rigid or flexible, pervious or impervious, seamed or seamless, wettable or nonwettable, and shaped into simple or branching tubes. The textiles employed are generally obtained from lots manufactured specifically for use as wearing apparel or household goods. In some instances the prostheses are fashioned by the manufacturer, but more often the home sewing machine is employed with the surgeon as seamstress. The experiments to test these prostheses are generally conducted as follows: The vascular substitute is implanted into the thoracic or abdominal aorta of dogs or pigs. These animals are then sacrificed at intervals (usually nearer one month than one year after transplantation) and the results, in terms of patency and histologic appearance, are reported. In some instances the material is given a limited clinical trial before the report is made. Occasionally the procedure is reversed and the vascular substitute is first tried clinically and then used in experimental animals!

There are several unique but unfortunate features of this type of investigation. First, the surgeon is in the difficult position of attempting to evaluate materials about which he knows almost nothing, since the technical aspects of textiles and plastics are not emphasized in medical curriculums! Second, it is extremely difficult to obtain separate lots of material with identical properties, particularly if they are procured from the processor rather than from the manufacturer. Thus, the results of one phase of the experiment may not be comparable with another, although materials of the same general construction are used. Finally, there is little attempt to exchange

observations except in formal reports, and here significant details are usually lacking. Occasionally, the surgeon may obtain from a colleague a small swatch of material "to try out," but rarely will he know its specifications and properties.

Undoubtedly, something of merit will come of these poorly controlled experiments, although at the moment their chief value appears to be the opportunity afforded surgical residents to become skilled in vascular anastomoses. Meanwhile, the day of the ideal substitute is postponed.

The solution to the problem of developing a satisfactory vascular substitute is obvious to many and consists of enlisting the technical knowledge and resources of industry. In fact, this step has already been taken by at least two investigators in the field. However, to be most effective, such a program should be conducted as a cooperative effort supported by all surgeons who are interested in vascular disorders. It would seem logical, therefore, for one of the surgical societies to appoint a committee to define the properties of an ideal vascular substitute. This committee should then secure the cooperation of the chemical and textile industries in the development of a suitable prosthesis. As materials are produced, they should be distributed to the various vascular centers for experimental and clinical trial. In this way, each material can be carefully evaluated within a relatively short time.

—Oscar Creech, Jr.

Appraisal of Progress in Surgical Therapy

CONDUCTED BY WARREN H. COLE, M.D.
ASSISTED BY
J. GARROTT ALLEN, M.D. CLARENCE DENNIS, M.D.

VASCULAR ACCIDENTS DURING PULMONARY RESECTION: PREVENTION AND CARE

DAVID V. PECORA, M.D., RAY BROOK, N. Y.

(From the Department of Surgery, Ray Brook State Tuberculosis Hospital)

IN SPITE of the many technical advances which have been made in the field of pulmonary surgery, references to severe hemorrhage occurring during pulmonary resection continue to appear in the literature. Although the subject has been discussed,[1-4] no presentation has appeared which offers a complete plan of management of the vascular accidents likely to be encountered. Moreover, personal experience has indicated that while many of the maneuvers described are of value, others are useless. It is the purpose of this paper to present a mode of approach which has been found exceedingly valuable.

The best preventative of vascular accidents is alertness to possible dangers and knowledge of proper precautionary measures. Although the great majority of resections offer no problem, there are occasional instances in which inflammatory changes or tumor make dissection extremely hazardous. Moreover, degenerative changes within the vessel wall, especially the pulmonary artery, may be accompanied by extreme degrees of friability. Under these circumstances, not only should one exercise the greatest gentleness, but, wherever feasible, one should dissect peripherally in order to deal with vessels of a minimum caliber. If, however, it should prove necessary to ligate a large vessel, contrary to the recommendations of many, only enough of the vessel to allow passage of a ligature should be freed. If bleeding occurs under these circumstances the opening can be plugged by packing with a substance such as Gelfoam and approximating the surrounding tissues with a fine stitch; or Allis clamps may be employed as will be described. Following this, dissection may proceed at a slightly different level. Once the vessel has been ligated the procedure is repeated distally, and the vessel is severed proximal to the distal ligature leaving the portion of the vessel between the point of transection and the proximal ligature adherent to the perivascular tissue. This maneuver not only exposes a minimum of the vessel wall to trauma, but it also provides a bridge of tissue distal to the proximal ligature which prevents the latter from slipping.

Although it has been emphasized[4] that dissection is facilitated if the perivascular sheath is followed, this may not be advisable in certain rare inflammatory conditions. Under these circumstances, if one persists in attempts to

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locate a sheath he may find remaining a very thin friable vessel consisting essentially of media and intima. Since bleeding from such vessels is extremely difficult to control, one would have a greater margin of safety by leaving a small amount of perivascular tissue attached to the vessel. In the case of pneumonectomy it may be safer to perform an intrapericardial dissection as discussed following.

When resection is performed for carcinoma it is rarely advisable to attempt to free vessels at a level involved by tumor. Under such circumstances any benefit which might be derived is far outweighed by the risks encountered. If it is impossible to carry the dissection to a more central level free of tumor, resection should be abandoned in favor of nonsurgical treatment such as roentgen therapy. Before abandoning resection, however, the pericardium should be opened as recommended by Allison.[6] One should never proceed with pneumonectomy until he has determined that all vessels are sufficiently free of tumor to

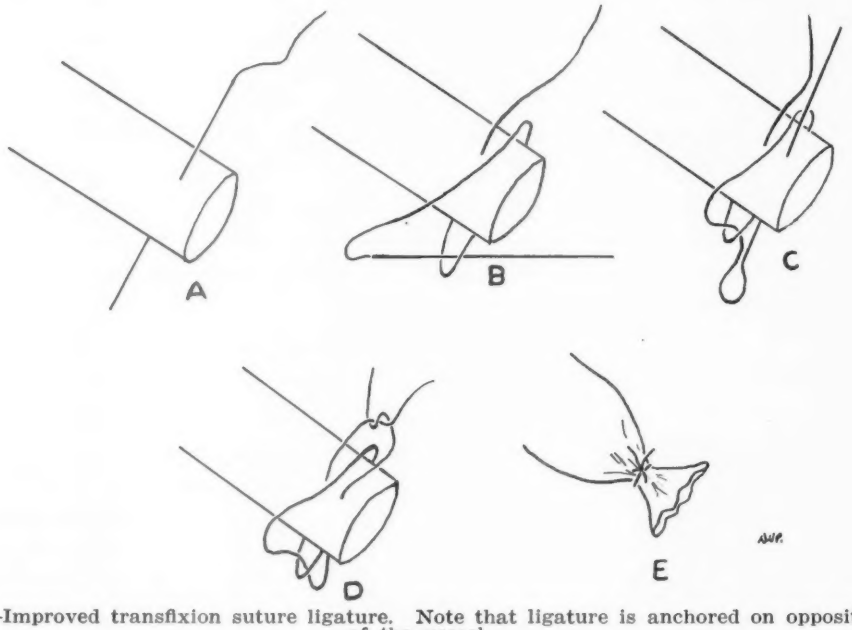

Fig. 1.—Improved transfixion suture ligature. Note that ligature is anchored on opposite sides of the vessel.

allow ligation. Occasionally, when one of the veins is completely involved, one may remove a portion of the left auricle.[5, 6] If this is done, it is safer to clamp the auricle with a suitable clamp such as that of Harken and to insert a row of interrupted mattress sutures of atraumatic 00 braided silk on the cardiac side of the expected line of division. The sutures are left long for traction. Following division of the auricle external to the first layer of sutures, a second layer of the same suture material is employed as an over-and-over continuous lock stitch external to the mattress sutures. If a layer of sutures is not inserted prior to incising the auricle one may find it difficult to close the auricle in the event that the clamp should slip. If it is necessary to leave a short venous or arterial stump after ligation, the end of the vessel should be oversewn with fine

silk, as recommended by Mendelsohn.[4] An over-and-over lock stitch is adequate. Procedures on the auricle or the vessels are facilitated if the other structures have been divided previously, thus facilitating early removal of the lung to provide an unobstructed field of vision.

Whenever it is necessary to divide the veins prior to the pulmonary artery, the latter vessel should be ligated first in continuity. This not only prevents loss of blood into the lung as has been emphasized,[2] but it also prevents distention of the organ, which at times can predispose to accidents by making exposure difficult.

Whenever possible a long cuff should be left distal to a ligature on a vessel to prevent slipping. Furthermore, ligation of an artery should be performed without too much tension as it is possible to fracture the vessel wall. Parenthetically it may be stated that veins show little tendency to fracture under similar circumstances. Some preparations of oiled silk ligatures tend to glide poorly when knotted, thus making it difficult for a surgeon to judge the actual tension transmitted to the loop about the vessel. Waxed silk seems to be more satisfactory in this respect. It is advisable before severing a vessel to make a small preliminary nick between ligatures; if bleeding is encountered one may then religate the vessel without fear of excessive blood loss.

Much use has been made of the transfixion suture ligature which passes once through the vessel wall after which it is tied on both sides. After a severe hemorrhage due to slipping of this type of suture ligature, a new one was devised (Fig. 1). Although the new suture-ligature appears superior, it is probably not as safe as an occluding distal suture of fine silk.

It has been suggested[4] that, in subtotal pulmonary resections when peripheral vascular injury is likely, the main pulmonary vessel(s) be dissected free so that proximal ligation may be performed in case of hemorrhage. Two objections have been found for this procedure: (1) it requires dissection of a larger vessel which in turn might be injured; and (2) it does not control "back bleeding" from the periphery of the lung. Use of the hilum tourniquet in such emergencies, as recommended by Soutter,[7] eliminates both these objections and, in addition, offers the advantages of speed and ease of application.

Prophylaxis against hemorrhage from the vessels in the main pulmonary hilum presents a much more difficult problem. If an accident is considered likely one should open the pericardium anterior to the phrenic nerve to facilitate the maneuvers described here.

Most surgeons employ digital pressure to initially control bleeding vessels. Although it has been stated that further dissection can be performed while digital pressure is maintained,[4] it is actually difficult to perform. In the control of hemorrhage personal experience has indicated that Allis (or Judd-Allis) clamps may be employed as shown in Fig. 2 to approximate the edges of the vascular laceration as the occluding finger is slowly removed. If the bleeding vessel is small, one clamp is sufficient. If the vascular rent is large, several clamps may be applied a few millimeters apart. If necessary, one or more clamps may be removed and reapplied individually with only minimal bleeding. The vessel is then sutured by a continuous lock stitch of suitable silk as demonstrated in

Fig. 2. Note that the suture is passed beneath the clamp and locked before the clamp is removed. The end of the right main stem pulmonary artery has been successfully sutured in this manner. Usually only one layer of suture material is necessary.

If the wall of a vessel is fractured, it is usually not necessary to place an occluding clamp upon the vessel. Indeed, this might be dangerous. If bleeding is absent or minimal the edges of the fracture are approximated by a continuous over-and-over lock stitch of 5-0 arterial silk through all layers. The small amount of bleeding which occurs about the needle perforations soon stops spontaneously, if suture is carefully performed.

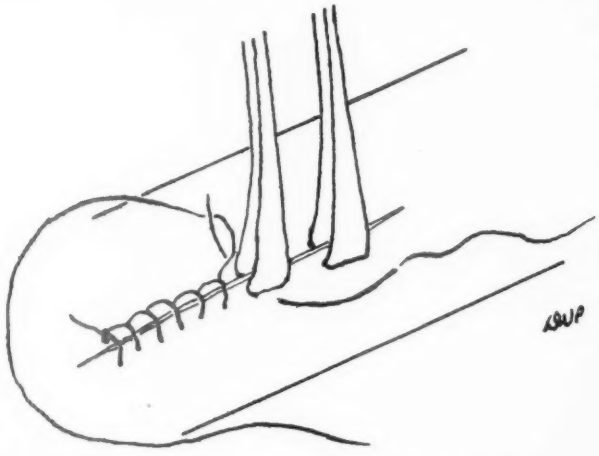

Fig. 2.—Method of suturing a rent in a vessel. Allis clamps have been applied and are removed as suturing progresses. A continuous lock stitch is employed. Note that a stitch is taken beneath the clamp before the latter is removed.

If one should encounter from one of the main pulmonary vessels hemorrhage which is uncontrollable by this method, the pericardium should be opened anterior to the phrenic nerve. Although it has been found necessary personally to stop bleeding by intrapericardial pressure only once (employing pressure as shown in Fig. 3, *A* to control bleeding from the right pulmonary artery), this proved eminently successful. The following maneuvers, based on the anatomic studies of Allison[6] and Healey and Gibbon,[5] would appear to be equally sound. If one of the pulmonary veins is involved it is possible to compresss it intrapericardially between the thumb and forefinger. The distal end of the vein may then be occluded by suitable clamps (such as Allis), after which the vein may be ligated intrapericardially or its end sutured. If the right pulmonary artery is the vessel involved, intrapericardial pressure with the index finger at points *A* or *B* (Fig. 3) will control bleeding. Thereafter, the end of the artery is clamped as in the case of a vein. The artery may then be ligated in the transverse sinus as described by Healey and Gibbon[5] or its end may be sutured. Bleeding from the left pulmonary artery may be controlled by digital pressure at the point indicated by *C* in Fig. 4. Since it is difficult to ligate a short left pulmonary artery it would seem preferable to suture the end of the vessel after occlusion with Allis clamps.

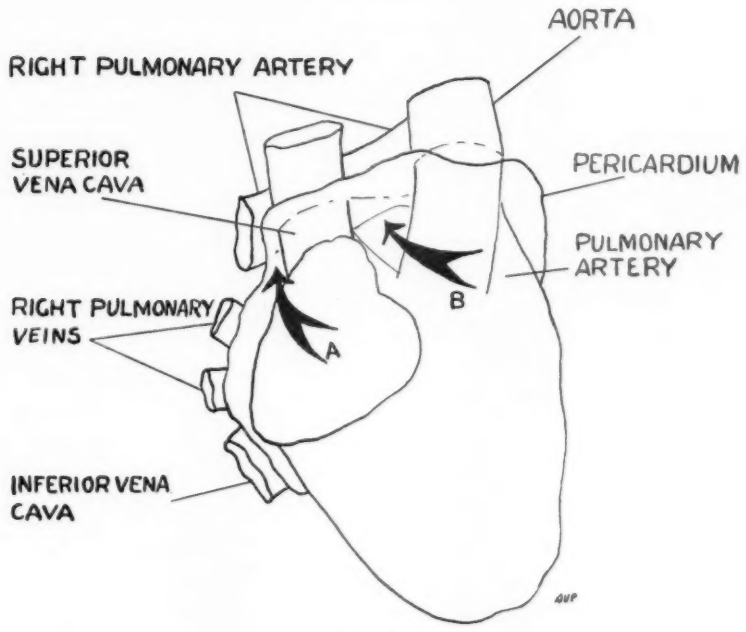

Fig. 3.

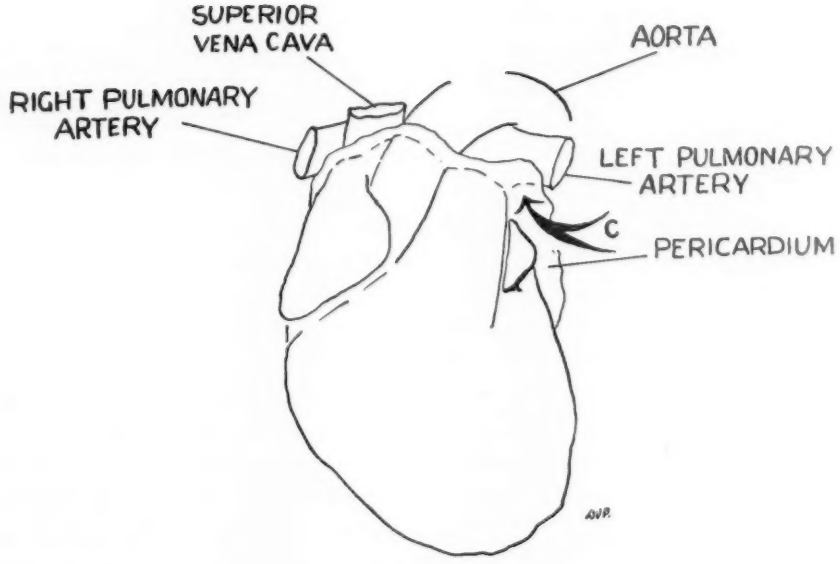

Fig. 4.

Figs. 3 and 4.—Diagrams of the heart and great vessels (after Sabotta). *A* and *B* (postcaval recess[4] and transverse sinus, respectively) where intrapericardial pressure may be employed to contral bleeding from the extrapericardial portion of the right pulmonary artery. Similarly, *C* represents the left pulmonary recess[6] where intrapericardial pressure may be employed to control bleeding from the extrapericardial portion of the left pulmonary artery.

SUMMARY

Prevention and care of vascular accidents have been discussed.

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ANNOUNCEMENT

The Horse Shoe Club

A program of exchange of surgical residents between this country and Great Britain has long been encouraged by the Horse Shoe Club. Arrangements for the exchange will be accomplished by the Graduate Training Committee of the American College of Surgeons in this country. The original plans call for an exchange of about one year's duration, the American resident usually being at the second or third year level. Those coming from Great Britain may be somewhat farther along in their training programs but would be fitted into the American residency so as to provide the best opportunity for training and experience. The host institution is expected to provide the living expenses of the visiting student, but the travel expense is to be borne by the parent hospital.

The Horse Shoe Club is composed of British, Canadian, and American members and was founded in 1932 for the purpose of fostering friendship and the exchange of medical, research, and clinical workers among the three countries. In Great Britain, the Club offers hospitality to visitors from the United States and Canada and assists in making arrangements for visits to British hospitals.

It is hoped that those who are responsible for approved programs of training in general surgery and the surgical specialties in this country will consider the possibility of participation in this exchange program for the mutual benefit of members of the profession in Great Britain and the United States. Specific inquiry concerning the program should be addressed to the American College of Surgeons. It should be emphasized that this program does not interfere in any way with exchange programs that now exist or that may be established. Private arrangements made between schools and hospitals in both countries are all important and it is hoped that they will be amplified. Under these circumstances the Horse Shoe Club offers friendship and aid when needed to the Fellows in both countries.

Correspondence

To the editor:

The following observations concerning the patient whose case was reported in the June, 1952, issue of SURGERY under the title "Aneurysm of the Hepatic Artery," seem of sufficient interest to be recorded.

The patient, a 57-year-old man, who had had four episodes of massive gastrointestinal hemorrhage, was treated by wiring an aneurysm of the hepatic artery which was believed to be the source of the bleeding. During the following eighteen months he remained asymptomatic save for two episodes of melena.

Then another massive gastrointestinal hemorrhage necessitated re-exploration. The proximal hepatic artery was pulsating, but the aneurysm and the hepatic artery distal to it felt solid and were not pulsating. When the aneurysm was separated from the duodenum, hemorrhage occurred from a tear in its wall. Most of the sac was filled with clot, but a channel remained from the point at which the hepatic artery entered the aneurysm, extending to the exit of the gastroduodenal artery where the duodenum was in close contact with the aneurysm. This vessel was pulsating and was ligated and divided. The hepatic artery was ligated in continuity. Because the presence of a duodenal ulcer was suspected, a partial gastrectomy was performed. The wall of the removed portion of duodenum on microscopic examination was found to have been replaced by fibroplastic proliferation and organizing hemorrhage, but the mucosa was intact.

The patient made an uneventful convalescence and has remained well for three years. At present, his liver is palpable 3 cm. below the right costal margin. All liver function studies are within normal limits except for the alkaline phosphatase which is 11.6 Bodansky units.

The second operation established that the patient's gastrointestinal hemorrhage was due to rupture of the hepatic aneurysm into the duodenum. Wiring of the aneurysm failed to occlude the gastroduodenal artery which was involved in the aneurysm. Ligation of this vessel and of the hepatic artery proximally has prevented further hemorrhage.

—*Richard W. Dwight, M.D.*
Boston, Mass.

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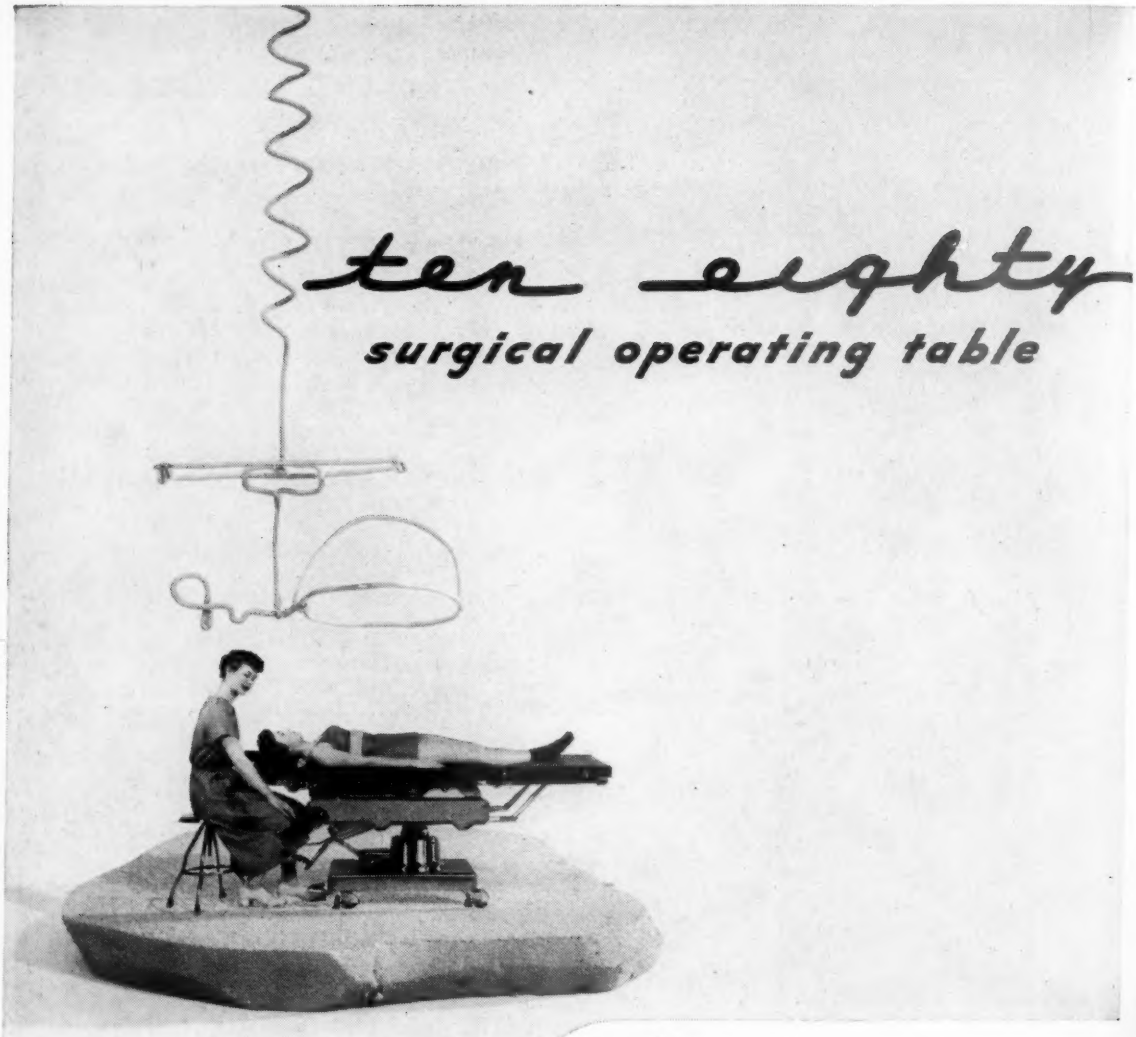

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1. Parsons, W. H., et al.: *Ann. Surg.* 135:791 *(June) 1952. 2. Whiting, J. A., and Hotz, R.: Surg., Gynec. & Obst.* 97:709 *(Dec.) 1953. 3. Beling, C. A.,* et al.: *Geriatrics* 7:179 *(May-June) 1952. 4. Barbour, C. M., Jr., and Tennant, R.: J. Urol.* 71:497 *(April) 1954.*

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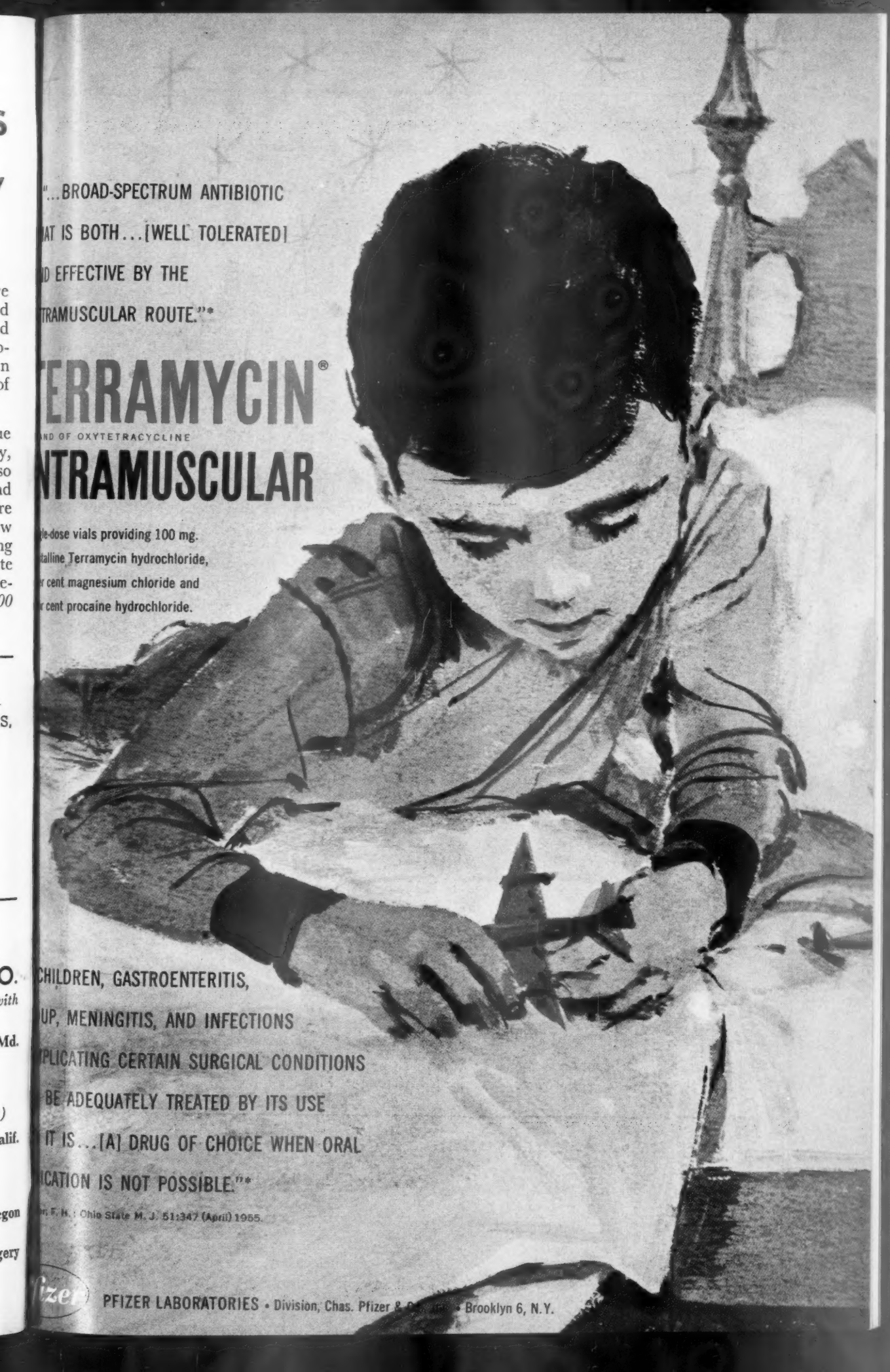
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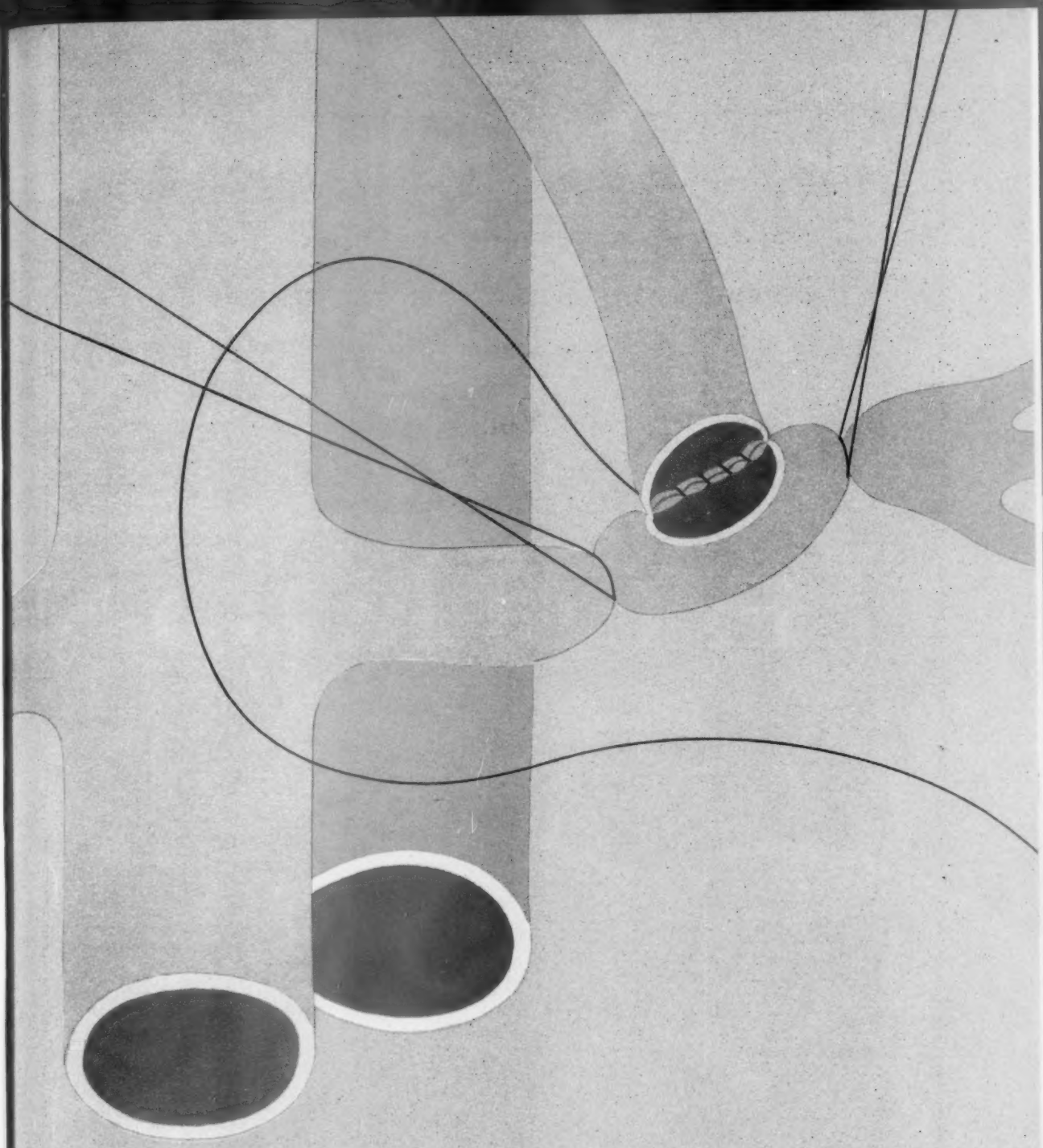

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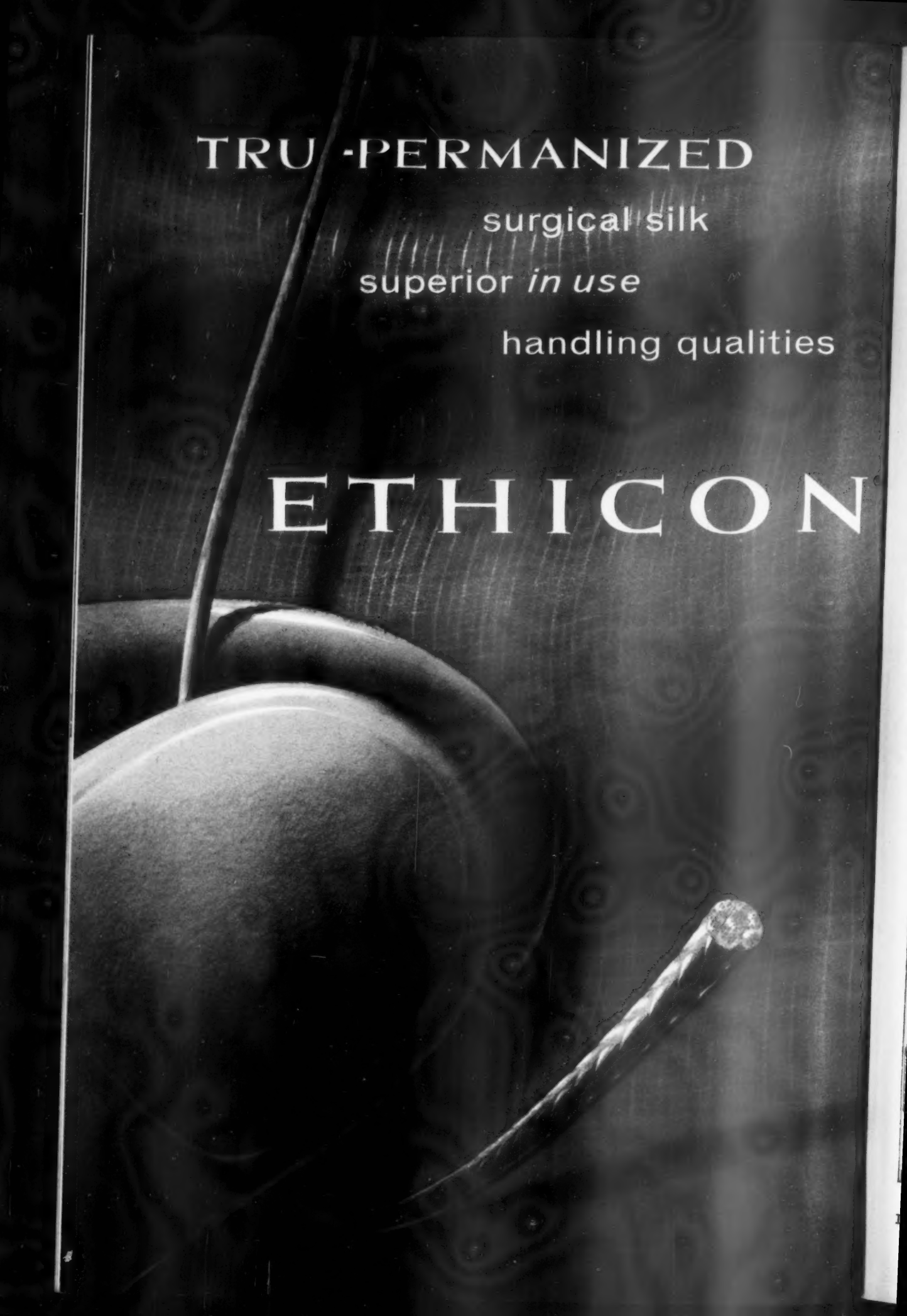
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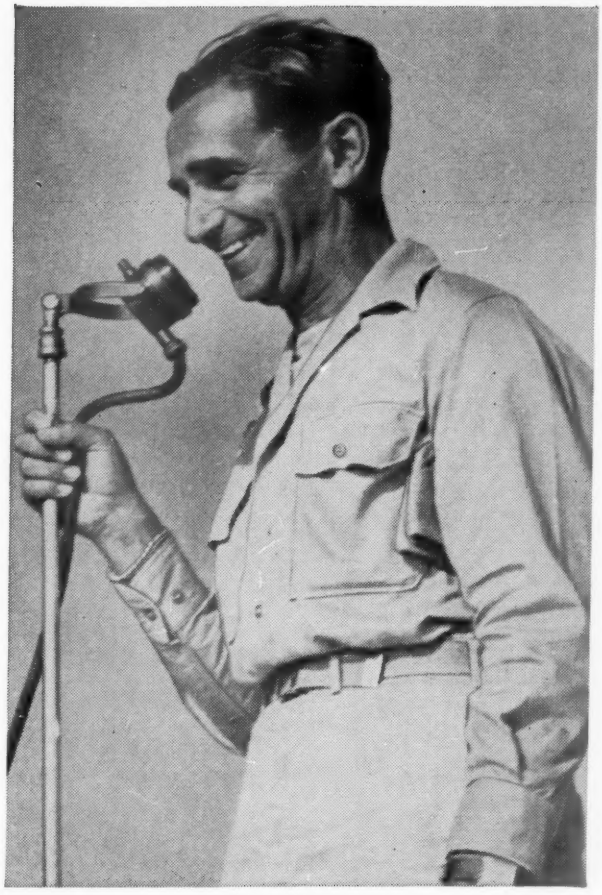

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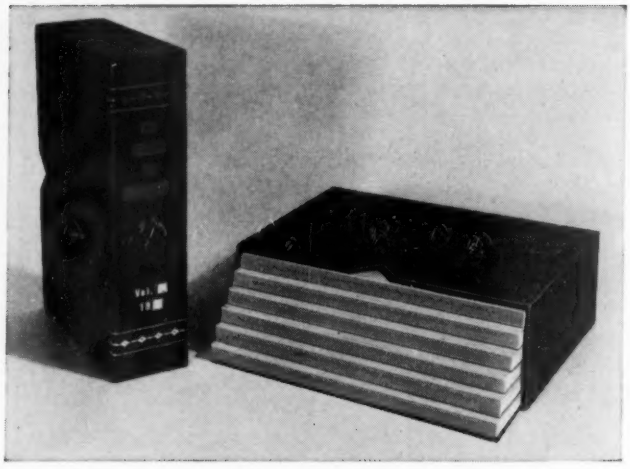

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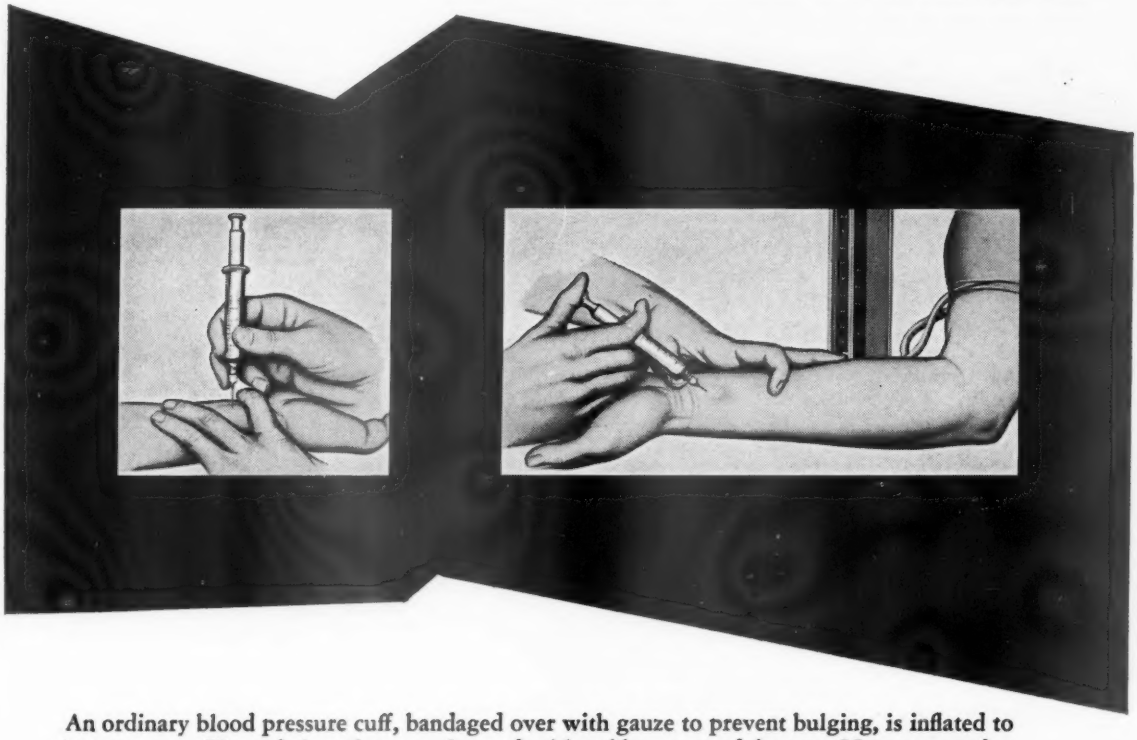

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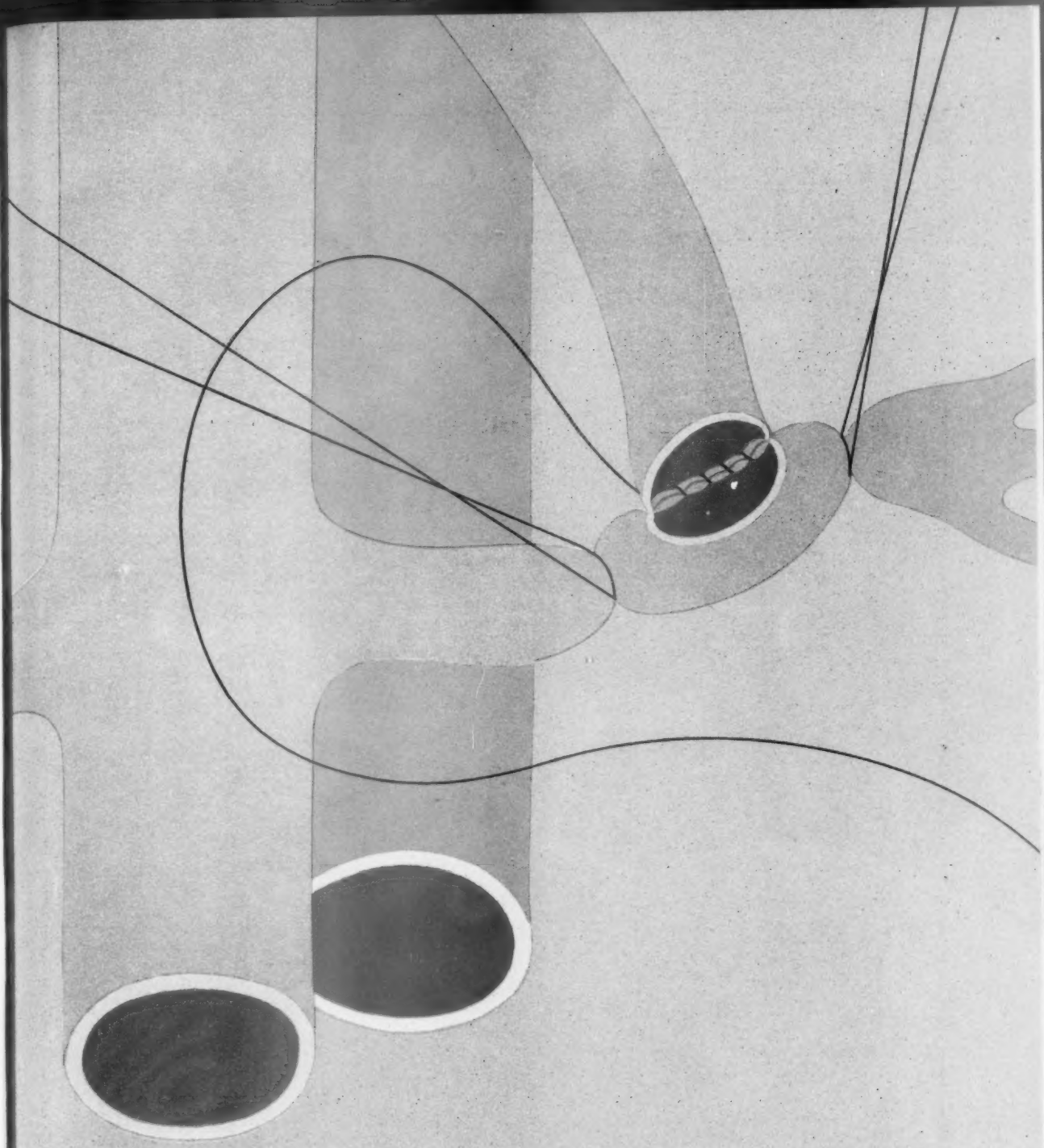

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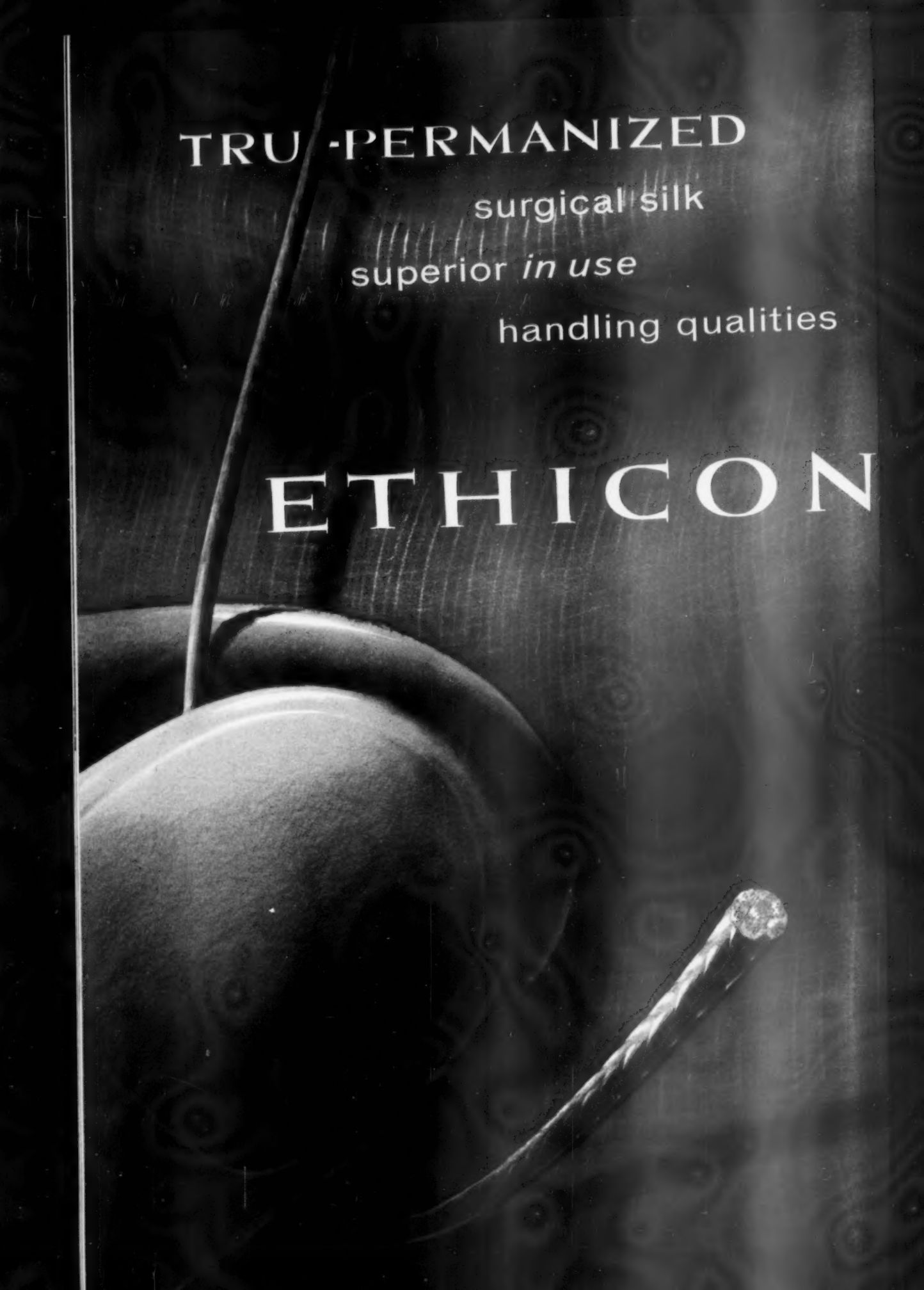
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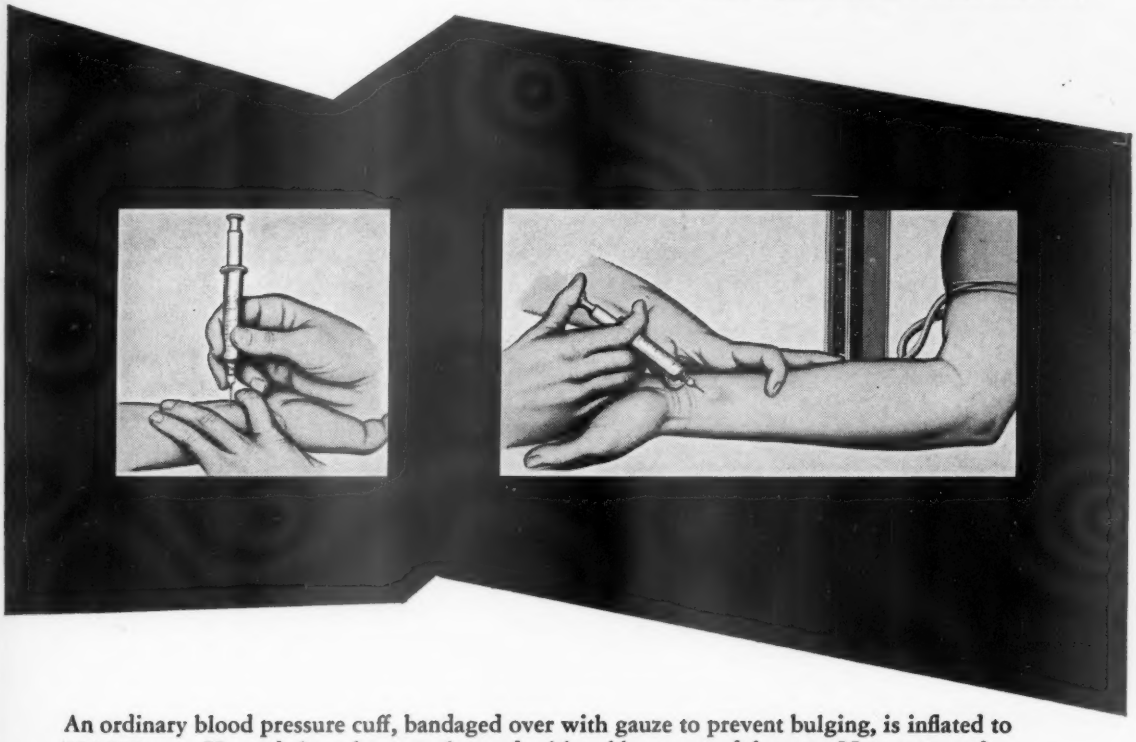

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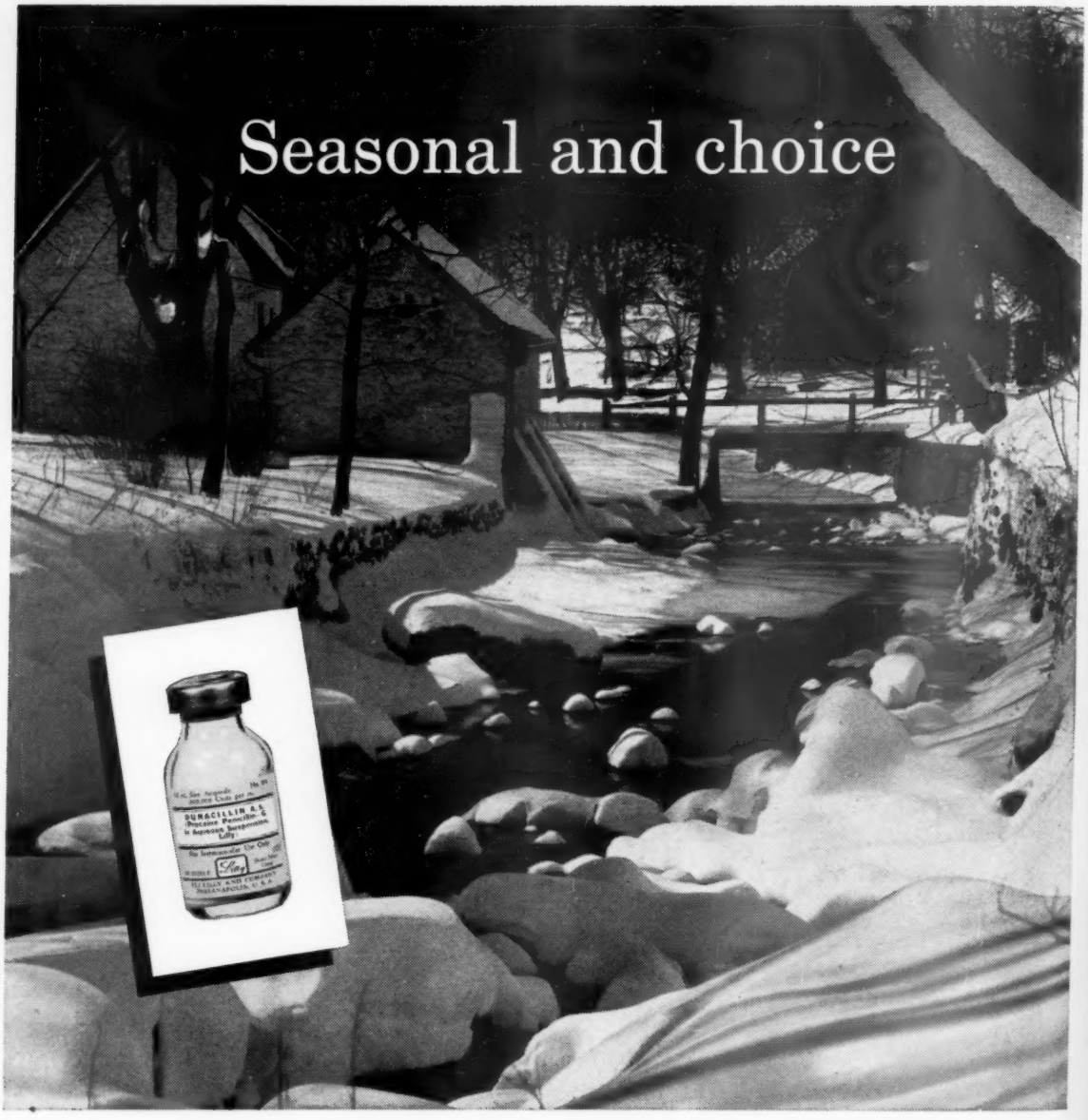

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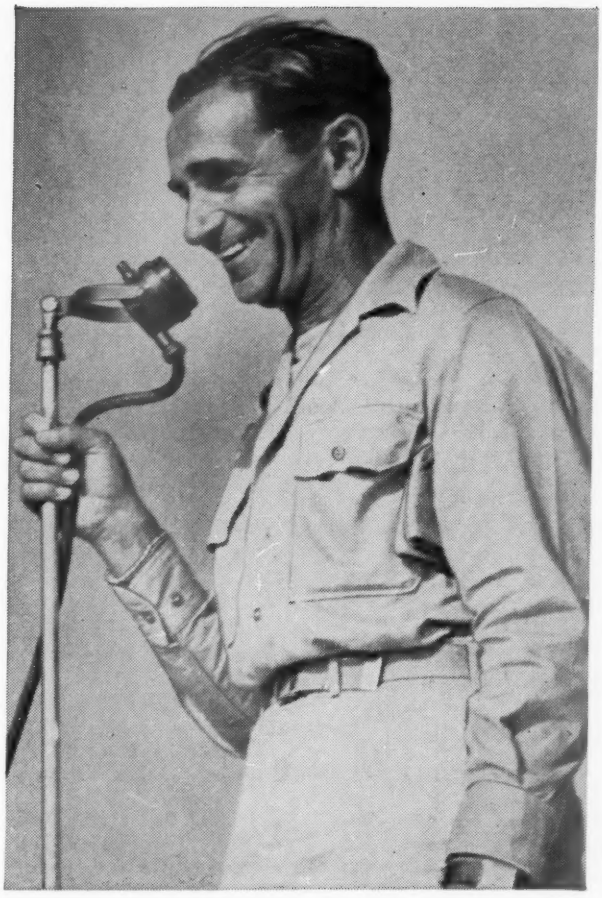

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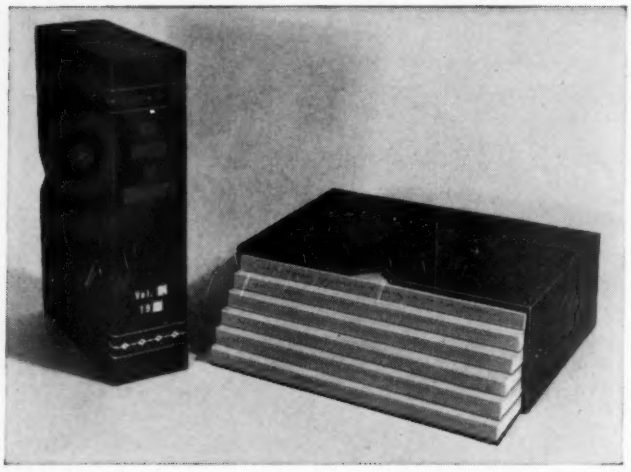

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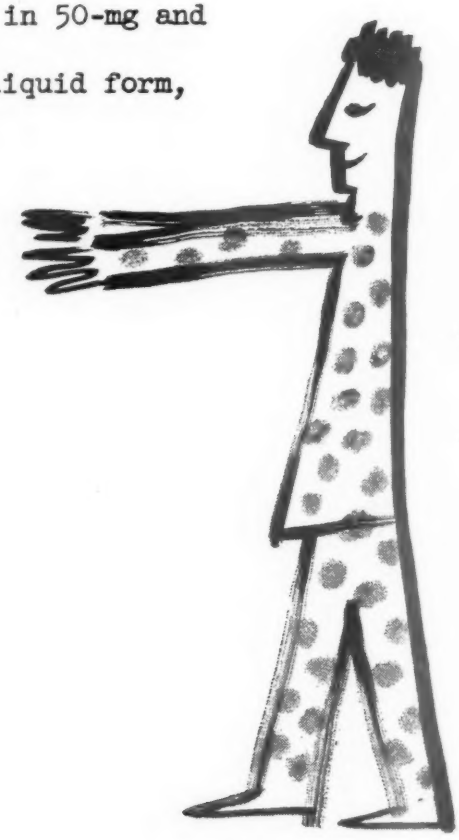
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